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PRESENT-DAY CONCEPTS OF EPILEPSY.¹

By E. GRAEME ROBERTSON,
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I AM grateful for the opportunity of commencing this symposium on a subject which has surely claimed attention since the dawn of reason and is still of paramount interest.

HISTORICAL SURVEY.

The ancient appellations, falling sickness, seizure, sacred disease, connote the early conceptions of epilepsy and its origin. Although some Hellenic physicians believed that diseases of the brain or other organs might provoke epilepsy, it was usually regarded as a disease in its own right. This view disappeared in the Middle Ages, when the unfortunate epileptic was regarded, and treated, as being possessed. With the Renaissance, inquiring minds once again examined the problems of epilepsy, and conceptions gradually emerged which flowered forth in the genius of Hughlings Jackson. He and his contemporary Sir William Gowers provided definitions of the term which, taken together, say much about epilepsy. Even if they are not definitions in the truest sense (for who can define

without full knowledge?) they have hardly been bettered since.

Gowers, the observant neurologist, defined the condition as "the recurrence of sudden brief disturbances of some function of the brain, varying in degree, extent and character, but generally attended with an arrest of consciousness sufficient, at least, to interrupt the control of muscles necessary for the maintenance of the erect posture". Hughlings Jackson, of philosophical bent, delved further into causation and considered "epilepsies on the hypothesis that the paroxysm is dependent on a sudden, temporary, excessive, disorderly discharge of some highly unstable region of the cerebral cortex".

Jackson's clear description of the form of epilepsy which came to bear his name was one of the greatest advances of all time in this field. He believed that an unstable part, anywhere in the cortex, could "store up" energy, and when it reached a certain degree of instability discharge might be provoked. Of attacks commencing in the motor cortex he made the following statement: "The march of the attack, the order in which the different parts of the body become involved, reveals the arrangement of the corresponding foci in the precentral region." He described spreading sensory epilepsies, and asserted that the function of the part of the brain in which a localized attack arose determined the content of the attack. An hallucination of any sense might be an epilepsy; and the qualities of the epileptic hallucinations were specific. "A

¹Read at a meeting of the Section of Neurology and Psychiatry, Australasian Medical Congress (British Medical Association), Eighth Session, Melbourne, August, 1952.

paroxysm of subjective sensation of smell is an epilepsy as much as is a paroxysm of convulsion." He was extremely interested in the epilepsy which originated in the temporal lobe, with its "turning inwards of consciousness" (Gowers), its vivid recall of a scene or situation which may seem very familiar and its hallucinations of taste and smell, often accompanied by chewing movements and salivation. In spite of the vivid nature of the hallucinosis, it is only vaguely integrated into memory. There is loss of consciousness of the actual environment, yet automatic activity may continue.

Pathological examination of causes of spreading epilepsies showed Jackson that space-occupying causes could initiate attacks, and allowed him to map out the arrangement of neurons in the motor and sensory cortex in terms of bodily function. In 1873 Ferrier demonstrated that movements could be produced by electrical stimulation of the exposed cortex of animals, and was able to ratify the earlier map of the cortex constructed from clinical data by his friend Jackson.

Thus it became apparent that epilepsy might be symptomatic and the idea grew that epilepsy was always a symptom and not a disease *sui generis*. However, it was considered that an hereditary factor was responsible in most instances, but the faith of the past that this was always so (which still lingers in the lay mind) was shaken.

MODERN RESEARCH.

Thus a new era opened with the belief that an inborn tendency to convulsions existed and that when it was present attacks might be precipitated by a number of causes.

Inheritance: Study of Twins.

The influence of heredity has been studied extensively, but never more simply than in the study of twins. Recently (1947) Lennox has studied 66 pairs of twins, one or both of which were subject to epilepsy. Forty-three pairs were monozygotic and 23 dizygotic. He found that both monozygotic twins were affected in a high percentage of cases, whereas both dizygotic twins were never affected. Statistical analysis suggested that predisposition and acquired pathological lesions were forces about equal in strength in the production of epilepsy.

Electroencephalography.

In 1928 Berger recorded action currents from the cerebral cortex. Since then a laboratory instrument has become an investigational tool and much of our new knowledge emanates from it.

Hereditary Transmission.

Electroencephalography has shown abnormal cerebral rhythms in a higher proportion of non-epileptic relatives of epileptics than in normal families. Lennox, Gibbs and Gibbs (1940) found abnormal records in 54% of parents, siblings and children of epileptics, compared with 6% without epileptic relatives. Löwenbach found 50% of healthy relatives of epileptics were affected. It should be stressed that in most cases the records were not specific of epilepsy.

The former authors believe that the dysrhythmia of epilepsy—predisposing to epileptic manifestations—is inheritable. Löwenbach believes that the abnormal records indicate a non-specific inherited instability of the nervous system which may, under certain circumstances, result in epilepsy.

Mechanisms of Epilepsy.

Much has been added. The analyses of wave forms made by Gray Walter are of such theoretical importance that mention must be made of them; yet it is impossible to deal with them in a paper of this scope. Gray Walter believes, in short, that certain epileptic conditions result from the synchronization of persistent quasi-harmonic cerebral rhythms.

Until recently it was held that epilepsy resulted at the cortical level. However, Morison and Dempsey (1942) showed that stimulation of the intralaminar portions of the thalamus produced a generalized bilateral response over the cerebral cortex. They postulated that a thalamo-cortical system controlled the spontaneous rhythmic activity of the cortex, various parts of this system controlling their own specific cortical regions. Jasper and Fortuyn (1947) were able to produce the wave-form of *petit mal* by rhythmical electrical stimulation of this part of the thalamus. Penfield and Jasper (1947) have extended the matter into a theory which postulates that abnormal activity of this region (which they believe to be responsible for consciousness) produces immediate loss of consciousness and the sudden appearance of wave and spike activity synchronously over the whole cortex, or in corresponding areas of each hemisphere. This dysrhythmia may depend upon inherited factors, for *petit mal* has never been shown to depend upon an organic lesion. Further, the authors believe that cortical lesions may secondarily stimulate discharges from this thalamic region. However, in the present state of knowledge it cannot be denied that cortical abnormality may cause local instability, which in turn may lead to excessive discharge and activate other cortical areas.

Types of Paroxysmal Epileptic Dysrhythmia.

Recording during attacks has shown that different types of attacks have clearly defined electroencephalographic counterparts.

1. *Petit mal* is associated with slow wave (three cycles per second) and rapid spike patterns repeated for some seconds. The recognition of *petit mal*, clinically or electroencephalographically, carries this important implication: search for a removable lesion is unnecessary, for none is known to cause *petit mal*.

2. *Grand mal* is associated with initial slowing of the record, with later superimposed fast activity. Two to seven per second waves appear and gradually increase in amplitude. The tonic stage is associated with the superimposition of rapid sharp waves (spikes) upon the slow rhythm. (Many of the spikes may be of muscular origin, but animal experiments suggest that 30 cycles per second activity is a cortical epileptic rhythm.) Later, the slow waves become slower and the spikes intermittent before finally disappearing. After the seizure random diffuse low voltage slow activity corresponds with the period of flaccidity or unconsciousness. After this automatic activity may supervene.

3. During Jacksonian attacks it may be possible to observe time intervals between the onset of a spreading dysrhythmia in the various tracings. The repetition of frequent attacks may allow of recording with modified electrode placements to demonstrate this.

4. Gibbs and Lennox (1938) and Gibbs and Gibbs (1941) described psychomotor seizures characterized by high voltage four to seven cycles per second activity.

5. A fifth type of attack is, according to Jasper, significant of a focal cerebral lesion and is associated with automatic activity. It consists of the rapid repetition of fast spikes, which may spread. This is found chiefly in the frontal and temporal regions, the type of automatism differing according to the site of origin.

Automatism.

An attempt to make the matter of epileptic automatism clearer may justify a digression. The following types may be recognized. (i) In the automatic behaviour which occurs after a major attack, the patient may be confused and resistive before full consciousness is regained. (ii) The subject of true *petit mal* may continue with unskilled activities during an attack. He may continue walking, although he usually stands still; he may continue to move his arms in coarse fashion, but cannot write. There is no hallucinatory content. Consciousness is expunged and no memory of the attack remains. (iii) In automatisms due to lesions of the frontal lobes the patient may become

temporarily confused and behaves in a fashion out of keeping with external circumstances. This may escape the notice of casual observers; for example, the patient may continue sitting in a tram, but travels beyond his stop if the attack outlasts his proper journey, or he may leave the tram in motion. (iv) In temporal lobe automatisms, having the qualities previous described (*déjà vu*, hallucinations of taste and smell *et cetera*), the patient shakes off any touch. The meekest individual violently resists interference, and he may wander away by himself. The epilepsy produces a sudden temporary change in his normal qualities. Temporarily he lacks judgement and insight, nor has he any memory of the episode afterwards. Removal of active temporal foci is being practised by a number of neurosurgeons, particularly Penfield.

Interictal Dysrhythmias.

Since it is uncommon to record the electrical activity of attacks, the occurrence of abnormalities between attacks and their significance become of great importance in diagnosis.

Wave-spike Patterns.—The form which is diagnostic of epilepsy is the occurrence of single wave and spike disturbances, or collections of insufficient duration to produce clinical attacks ("larval" forms of epilepsy). This does not necessarily indicate a diagnosis of *petit mal*, for the history may be one of major attacks. However, the patient may suffer from previously undetected minor attacks.

Random Spikes.—The occurrence of random spikes is, according to Jasper, the only form of electrical activity which characterizes a local epileptogenic lesion of the cortex. As has been indicated above, when the local spike discharge is repeated at a fairly rapid rate a change occurs in the behaviour and awareness of the patient. Spikes arising in the temporal lobe may be recorded only by electrodes applied to the base of the skull (pharyngeal electrodes).

Other Types of Dysrhythmia.—Many other types of dysrhythmia, fast and slow, exist. On the whole, it may be said that none are characteristic of epilepsy, although they may occur in epileptics. They may merely be an expression of a cerebral instability which may result in epilepsy, or of the cerebral deterioration accompanying epilepsy, or of the underlying pathological cause of the epilepsy. Obviously very careful evaluation is necessary, and some encephalographers are less conservative than others in assessment.

Encephalographic Diagnosis.

Absolute exclusion of the diagnosis of epilepsy can be made if an observed seizure has no accompanying dysrhythmia. Muscular movements may produce rapid spikes, but only the novice is likely to be deceived by the record. Thus hysteria may be recognized. If the patient refrains from having an attack, however, the diagnosis will remain obscure, for a normal record does not exclude epilepsy.

The diagnosis of epilepsy has usually to be made upon interseizure records, except in cases in which attacks of *petit mal* occur frequently. Walter and Dovey, in a survey of 2000 persons complaining of fits, found 52% of abnormal records. Other observers place abnormalities as high as 85%. Obviously there is hardly any evaluation more dependent upon individual standards.

Activation.

In order to increase the accuracy of diagnosis, a number of methods of "activation" have been elaborated, the accuracy of diagnosis, it is said, being increased to well nigh 100%.

The methods commonly used may be briefly described.

1. Hyperventilation is used as a routine procedure. Alkalosis and consequent cerebral vasoconstriction tend to increase the frequency of wave-spike patterns, but also increase the number of slow waves in normal young people.

2. If the attack is of reflex type, precipitated by some sensory stimulation (loud sounds *et cetera*), it is reasonable to attempt to reproduce this.

3. From early times it has been known that attacks occur during sleep and sometimes only during sleep. Gibbs and Gibbs state that sleep studies more than double the value of the electroencephalogram in diagnosis. Sleep may be natural or induced by "Seconal".

4. Photic stimulation is sometimes used. Walter, Dovey and Shipton (1946) consider that flashes of bright light repeated at certain frequencies tend to augment already existing frequencies, enlisting neighbouring frequencies into the augmented waves. This augmentation of frequencies already present may facilitate explosive epileptic discharges in predisposed subjects. At all events, attacks can be precipitated by flicker, and this may even occur clinically, as during a drive along a road with the sun shining between tree trunks on the patient's eyes, or it may sometimes be deliberately produced by the patient.

5. Subconvulsive doses of "Metrazol" given during recording may precipitate larval attacks.

6. Gastaut (1947) has shown that the most effective measure of all is the combined use of "Metrazol" and photic stimulation.

SUMMARY.

In summary it may be said that the electroencephalographic diagnosis of epilepsy may be made with certainty only when attacks during recording produce typical wave patterns. In most of these cases observation of the attack alone would establish the diagnosis quite as well. However, localizing evidence may be obtained, or the simultaneous appearance of wave-spike activity synchronously in both hemispheres suggests that further search for an acquired cause is unnecessary. Further, the information obtained may have a bearing upon treatment.

In most cases attacks do not occur during recording. Thus the clinical history of the patient and observation of an attack by a medical practitioner or (less reliable but more likely to be available) an eye-witness's account is more reliable than electroencephalography. This explains the sense of annoyance which arises in the mind of the neurologist when he is asked to make an electroencephalographic examination rather than to give an opinion upon the patient as a whole. A normal record does not exclude epilepsy, particularly if activation is not practised. Nor do all dysrhythmias clinch the diagnosis of epilepsy. However, certain types of dysrhythmia strongly favour the diagnosis of epilepsy (for example, larval attacks) and may serve to give warning that peculiar attacks are not functional. Finally, the value of the apparatus increases with the care of usage, the skill of recording and evaluation. Special methods are necessary to obtain maximal information.

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THE STRUCTURAL BASIS OF EPILEPSY.¹

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In this study we shall consider first to what extent epileptic attacks have a structural basis, by this being meant the extent to which significant pathological changes can be seen by the naked eye or by means of present-day histological techniques be found in the brains of those who have suffered epilepsy.

The answer to this is not convincingly given in the literature, and the incidence varies greatly with the source of the material. For instance, percentages derived from psychiatric institutes, from children's clinics or from service trainees belong to special groups, and even those obtained from the post-mortem room are suspect, for they deal with the dead alone, on whom of necessity some severe disease has operated. Indeed, unless some ruthless authoritarian investigator plans the destruction of a large cross-section of the epileptic population and its careful pathological examination, no precise answer is likely to be obtained.

However, one can determine a minimum percentage in a group of patients who have had epileptic attacks. This has been done for the purposes of this study in a series of 223 private patients, children and adults, examined by the one observer in the last few years. The selection has been alphabetical, and every subject who has had an epileptic attack has been included.

The methods of study have been the usual ones of history and clinical examination, with special methods of examination when possible. These have varied from case to case, according to the degree of suspicion of organic disease engendered in the mind of the examiner, to the inclinations of the patient, and also to the length of the hospital waiting lists. In almost all cases X-ray examination of the skull was carried out, in many encephalography or ventriculography with examination of the cerebro-spinal fluid, in a few angiography, and in a certain number electroencephalography, although this alone has not been used as conclusive evidence of organic disease.

The following were accepted as evidences of organic disease: such common features of organic disease as are shown by neurological examination; a history of severe head injury with unconsciousness followed by epilepsy; late epilepsy with severe arteriosclerotic changes; structural changes in the brain shown by plain X-ray examination, by air studies or by angiography; pronounced changes in the cerebro-spinal fluid—as, for instance, abnormal cells or a positive reaction to the Wassermann test. No doubt in a small minority of such cases the conclusion reached has been incorrect.

In the non-structural group were included such patients as had no clinical evidence of organic disease, and a family history of epilepsy; or those who with a long history of epilepsy from childhood were without clinical evidence of organic disease; or those in whom in particular air studies with adequate filling revealed no abnormality. However, all these deductions are liable to error, and I have no doubt that if every patient had been studied in full, structural disease would have been found in several of this group.

In an indefinite group were placed such cases as seemed suggestive of organic disease, but in which proof was lacking. These included patients who had suffered mild head injury or late epilepsy without evidence of disease, or who had focal attacks. Many of these are still awaiting admission to public hospitals; others have been admitted to a repatriation hospital, but the results of examination are not as yet forthcoming; others again had either refused or postponed investigation.

The result of this subdivision is as follows: proven structural change, 82 cases; no structural change, 93 cases; inconclusive cases, 48.

If we suspend judgement on the inconclusive group for a moment we have 82 organic cases as against 93 non-organic cases, in a total of 175 in which some conclusion has been come to. This is an incidence of organic cases of about 46%. But can we form some idea of the figure which might have been obtained if the inconclusive group as well had been fully studied?

Of these 48 cases air studies were unsatisfactory in three, and in three others the result was still inconclusive, 42 being left. As 76 air studies had been done in the entire group, this means that inconclusive information had been obtained from these in about 8%. But in the remaining 70 structural changes were found in 44, or nearly two in three. A similar incidence would in all probability have been obtained in the group of suspects who were awaiting investigation, or approximately 30 cases. If this number is added to the group already shown to be of organic origin, it would make a group of 112 or half of all cases. But even this percentage is probably too low, for organic disease is likely to have been present in several members of the group thought to have no structural changes.

However, one may sum up by saying that in those who have suffered epileptic attacks structural change was probably present in at least one in two.

The next matter to be considered is the incidence of particular pathological conditions in the causation of epilepsy. An analysis of the 79 proven cases in this group is as follows: trauma, 23 cases; vascular disease, 13 cases; cerebral tumour, seven cases; congenital deformations, three cases; syphilis, two cases; cerebral abscess, two cases; tuberculous sclerosis, one case; miscellaneous organic causes, nine cases; dilated ventricles of uncertain origin shown in air studies, 20 cases.

Trauma was the commonest lesion associated with epilepsy, occurring in 23 of the 79 cases; three cases were the result of birth injury. But even this high figure is too low, for it is probable that of the 20 cases in which dilated ventricles, unilateral or bilateral, of unknown origin were demonstrated by air study, several would have been the result of trauma sustained at some time from birth onwards. For injury to the brain commonly results in loss of white substance, from the removal of degenerated myelin, with compensatory dilatation of ventricles. The occurrence of trauma in early childhood may not be known and is frequently difficult to elicit, even in later life. Trauma must therefore be regarded as the commonest structural cause of epilepsy in the group under consideration.

Next in order of frequency comes vascular disease with 13 cases. But here again certain cases in which ventricular dilatation of unknown origin was demonstrated would certainly have a vascular origin, for profound vascular changes in the brain may be present without corresponding peripheral disease. The commonest cause was the vascular occlusion of adult life, and a first attack of epilepsy in middle age is likely to be of vascular origin.

There were seven proven cases of cerebral tumour in the group. This would make an incidence of 3.1% of the entire group of epileptics. Nevertheless, it is likely that even in the group of those deemed to have no organic disease an odd tumour may have remained unsuspected, and this percentage must be regarded as a minimum one; between 4% and 5% may be guessed at as rather more likely.

Congenital malformations of the brain—as, for instance microcephaly—were responsible for three cases. Syphilis (two cases) played a minor role, and epilepsy in these cases was the product of old infections. The scars left by treated brain abscesses were the exciting cause twice, and such diseases as cerebral aneurysm, tuberculous sclerosis and meningitis were responsible for epilepsy in single cases.

Summary.

In summary, in this unselected group of 223 epileptics of all ages, an organic focus was found in 82 or 36%;

¹Read at a meeting of the Section of Neurology and Psychiatry, Australasian Medical Congress (British Medical Association), Eighth Session, Melbourne, August, 1952.

but in addition there were 48 cases in which this disease was suspect but not proven, chiefly through insufficient examination. From comparison of a control series in which this had been undertaken, it is believed that at least one in two would have shown organic changes, which would make a minimum percentage of 47% for the entire group. Undoubtedly this figure would be too low, and it can be expected in any unselected group of epileptics of all ages that at least one in two would have epilepsy of organic origin. The value of a full investigation of all epileptics is then surely unquestionable.

Of the structural causes trauma proved the commonest, with vascular disease second and cerebral tumour third. Syphilis played a minor role.

Conclusion.

In conclusion it must be realized that the methods used in this study for proof of structural changes were coarse and incomplete. Could the human brain in all its complexity be unfolded for full examination, without doubt the percentage of structural changes found would have been greatly increased.

QUEER TURNS—THE LESSER KNOWN MANIFESTATIONS OF EPILEPSY.¹

By JOHN F. WILLIAMS,
Melbourne.

MANY patients complain of turns which are queer to them, and many turns seem queer to us, and those which are known as "*grand mal*" or "*petit mal*", the classical types of epileptic seizures, are of course very queer indeed. *Grand mal* was in fact so queer to the ancients that it was called the "sacred disease". This led to the famous Hippocratic statement: "It seems to me that the disease called sacred is no more divine than any other. Men think it divine merely because they do not understand it. But if they called everything divine which they did not understand, why there would be no end of divine things."

There are certainly many manifestations of these classical forms of epilepsy that we still do not understand, and they certainly are queer, in so far as little is known of their causation. This applies to the peculiar constitutional make-up of the patient subject to these turns, as well as to the particular sets of circumstances that lead to the sudden abnormal discharges of energy by the neurons, or the particular sets of neurons which discharge and cause the very wide variety of clinical appearances before, during and after the seizure.

This is not the paper in which to discuss all these difficulties, but it seems proper to mention some of the unusual features that are presented and to emphasize the difficulties in defining what is covered by the term epilepsy before embarking on a description of what may rightly be termed "the lesser known manifestations of epilepsy".

It seems that we must draw a distinction between those attacks which occur without known precipitating factors and those which require powerful stimulation of cells which, apart from such stimulation, show no abnormal discharges. An example of the latter can be found in the therapeutic convulsions induced by electrical or chemical stimuli for the relief of psychiatric disorders. These are often generalized convulsions, but at times, particularly with chemical stimulation from "Cardiazol", there was often to be seen nothing more than a momentary loss of consciousness, often associated with horrible feelings of impending dissolution and slight clonic movements. The variation in the results from apparently identical injections in different patients showed quite clearly the varying degrees of resis-

tance. The same lesson is shown in electrical stimulation, and it is also apparently true that at times when the patient is hovering on the brink of a convulsion, some extra stimulus, such as tapping the forehead, seems sufficient to tip the scales and bring about the desired result in an avalanche of neural discharge and a major convulsion.

It seems that we must recognize a gradient of susceptibility, at one end of which are those so susceptible to discharge that even when no turns or attacks are apparent and the patient is apparently resting quietly there are to be seen larval discharges in the electroencephalogram. These are the so-called idiopathic or cryptogenic epileptics. At the other are those resistant to very powerful electrical or chemical stimulation and with no signs of cortical dysrhythmia in the electroencephalogram.

If we attempt to limit the term epilepsy to those showing dysrhythmia in the electroencephalogram, it appears that we have to say that there are 15% of those with clinical forms of epilepsy who show no such findings in the intervals between attacks, and that 10% of the population show abnormal readings and that only 1 in 20 of these have seizures.

How, then, can we say whether any queer turn is epileptic or not? And are we to talk of asymptomatic epilepsy in those with no other signs than abnormal cerebral rhythms? Surely that would be a very queer turn indeed.

Let us turn to the clinical features of epileptic attacks.

It is generally accepted that turns of varying severity, which nevertheless appear to be epileptic in nature, can be precipitated by external stimuli. Numerous cases have been reported in which only music, generally classical as opposed to jazz, precipitates attacks which vary from temporary loss of consciousness and pallor to typical *grand mal*. Cerebral dysrhythmia has been reported in these in the interparoxysmal as well as the paroxysmal states. It appears that the emotional state engendered by the music plays an important role, and at times the fit can be prevented when the typical emotional state is developing by turning off the music. A local lesion in the temporal lobe has been found in several such patients.

However, attacks may also be precipitated by repetition of non-musical sounds or a sudden loud noise, as well as by strong visual stimulation of a certain frequency—photogenic epilepsy. Such attacks are often not affected by anticonvulsant drugs.

Kinnier Wilson quotes many other reflex forms of epilepsy, amongst the stimuli that precipitated the attacks being an unexpected touch on head or face (Hughlings Jackson) and pulling the stocking off the right leg of a little boy. It is found that the unexpected nature of the stimulus appears important in precipitation of the attack; but it is also found that in some cases a powerful sharp stimulus will check an impending attack—for example, tightening of a ligature in a case of Jacksonian epilepsy commencing in one limb. Mental or physical effort also has an inhibiting effect.

When attacks are precipitated by an emotional situation, suspicion is aroused that they are hysterical rather than epileptic; but such is not always the case. Some originally thought to be hysterical have been later found to be organic in origin.

There is no reason why a patient with epileptic attacks should not also have hysterical attacks; but the term hystero-epilepsy seems rather to beg the question of aetiology and diagnosis. Similarly, an hysterical person with the fundamental suggestibility characteristic of hysterics may, after many medical interviews and the inevitable inquiries as to whether he has bitten his tongue or wet his clothes, or after having seen an epileptic convulsion, produce an attack with many of the appearances of true epilepsy.

The difficulties of diagnosis are enhanced when, as is often the case, the attacks are not seen and reliance has to be placed on the accounts of relatives and onlookers; but the distinction between hysterical and epileptic seizures is generally clear.

¹Read at a meeting of the Section of Neurology and Psychiatry, Australasian Medical Congress (British Medical Association), Eighth Session, Melbourne, August, 1952.

It can, at any rate, be stated that precipitation of an attack by external stimulation of a physical or emotional nature does not mean that the attack is not epileptic.

It may be emphasized that the typical epileptic attack does not exist, for many variations can occur even within undoubtedly epileptic attacks.

Amongst the lesser known manifestations we may perhaps mention some of the aura and their importance as localizing signs of cortical damage which may not be detected in other ways. Aura are generally specific for the individual case, and may include changes in somatic, visceral or special sensibility. Amongst the first may be mentioned feelings that the limbs are moving, and amongst the second, the well-known epigastric sensation. Aura also include more complex states, such as the dream states, *déjà vu* phenomena, feelings of strangeness and unreality, horrible thoughts and particularly horrible smells, ill-defined fears, apprehension and terror—all of which may last for a few minutes. Like all the auras, these may occur alone or be followed by a generalized convulsion. This may, of course, lead to the forgetting of an aura which we may presume to have been present.

In major attacks we may also mention the post-convulsive phenomena, which also vary widely. Some patients feel better, as if some harmful influence that had been building up for days, as shown in the prodromata, had been removed by the convulsion. This effect has been stressed in years gone by by Muskens (as evidence of detoxication). Others, however, feel worse, with depression and exhaustion. Some return to normality quickly, others slowly, with a period of confusion in which violent crimes may be committed or apparently normal behaviour occur, which is later forgotten—the so-called fugues or automatisms.

These post-convulsive phenomena may also occur after minor attacks, which may not be diagnosed for many years and are then brought to notice only by the occurrence of a major attack; but whether these ever occur alone as "equivalents" is doubtful. It is tempting to explain much antisocial behaviour, both in children and in adults, as being due to masked epilepsy and cerebral dysrhythmia, and some supporting figures have been quoted by Jasper and others in this regard. Caution is necessary, particularly in regard to children, whose cerebral rhythm is still immature and unstable, and to quote Lennox, "gross dysrhythmia is consistent with normal personality". Maudsley in 1870 stated that "children of 3 or 4 are sometimes seized with attacks of violent shrieking, desperate stubbornness or furious rage, when they bite, kick and do all the destruction they can; these seizures, which are a sort of vicarious epilepsy, come on periodically and either pass in the course of a few months into regular epilepsy or may alternate with it". Maudsley also states that "when ever a murder has been committed suddenly, without premeditation, without malice, without motive, openly, and in a way quite different from the way in which murders are commonly done, we ought to look carefully for evidence of previous epilepsy, and other symptoms allied to epilepsy". (These statements of Maudsley are quoted by Lennox in "Personality and the Behaviour Disorder".)

It seems that we should divide minor attacks into "*petit mal*" and "minor motor seizures" as well as other forms. *Petit mal* is shown by sudden short attacks of staring that may occur very frequently. There is minimal motor activity; but major attacks may also occur either at the onset or after puberty. Other subjects (10%) seem to lose their seizures at puberty. *Petit mal* states may occur with the appearance of semiconsciousness and confusion and lead to a diagnosis of equivalents or mental confusion, but as a rule there are few after-effects. These *petit mal* attacks are not relieved as a rule by phenobarbital or "Dilantin", but are relieved by "Tridione". They are readily induced by hyperventilation and occur in children of a rather different type from those who suffer from the convulsive form; they are often rather frail, quiet and inactive, with good intelligence and a sense of ambition and responsibility. Amongst this group are to be found those formerly regarded as suffering from "pyknolespy"—

that is, a separate form of seizure with "myriad spells" and a good prognosis for cessation of attacks at puberty. It seems that this separation cannot be wholly justified.

"Minor motor seizures" are regarded as more common than true *petit mal*, and these appear to be abortive forms of a major attack with similar aura and early phases. Consciousness is lost to a variable degree, and after-effects are generally more pronounced. Reaction to phenobarbital and "Dilantin" is more often satisfactory and "Tridione" less often satisfactory in these than in *petit mal*.

Other minor attacks are called akinetic and are characterized by sudden losses of postural tone so that the patient suddenly crumples up, either wholly with sudden falls or partially with sudden falling of the head. They are also called inhibitory attacks and resemble the cataplexy associated with narcolepsy, to be mentioned later.

Myoclonic seizures also occur as minor attacks. Their frequency has been disputed, for while Muskens emphasized their importance and put their frequency at 50% of all epileptics, others have found a much smaller percentage (Bridge, 12% in children). They may occur as the sole epileptic sign, or they may appear as prodromata preceding the major convulsions or occurring in the intervals between them. They generally affect one region—for example, the arms—and the jerks may be such that articles are thrown, the hand is jerked back or the patient is thrown to the floor. Consciousness is very little affected. These attacks appear to occur most often in the morning and may be a danger during the morning shave, but they may also occur throughout the day.

There is another rather rare type of minor attack which consists of almost continuous twitching movements of a portion of a limb rather than formed movements—the so-called "*epilepsia partialis continua*"—while on the other hand there are attacks in which the movements are coordinated and semi-purposive rather than convulsive, and yet seem to be epileptic rather than hysterical. There are others again in which there are only slow tonic contractions associated at times with loss of consciousness and micturition.

There is also a group described by Kinnier Wilson as "periventricular epilepsy", wherein are to be found Gowers' vasovagal attacks. They may be shown by sudden changes in the heart beat, or in breathing, and associated with feelings of choking, a turning over of the stomach, hot flushes or icy chills, pallor, sweating, shivering and tremors, as well as feelings of fear, agony or impending dissolution. These are, of course, many of the symptoms which occur in a state of acute anxiety, and the differentiation may depend on the setting in which they occur rather than on the symptoms themselves. Syncopal attacks may also apparently arise on a neural rather than on a circulatory basis, while attacks of yawning and rises of temperature have also been ascribed to epilepsy.

Some of these attacks seem far removed from our usual conceptions of epilepsy; but it seems that cerebral dysrhythmia is present at times in most if not all of them, and if this is made the distinguishing feature of epilepsy, epileptic they must be.

There are, however, other groups of seizures which show no evidence of dysrhythmia, but which in other ways resemble epileptic seizures. Narcoleptic attacks with fits of sleep of varying depth and transient attacks of tonelessness or powerlessness, so-called cataplexy, is one such group. The latter, cataplexy, generally follows an emotional experience such as one causing anger, annoyance, anxiety, joy or laughter, which must be irresistible, while one patient is quoted by Kinnier Wilson whose attacks occurred at the moment of sexual orgasm and another whose first attack occurred when he hooked a fish. They may be associated with symptoms resembling vasovagal attacks. Some narcoleptic patients pass into trance-like "cataleptic" states in which they are aware of their surroundings, but are unable to move, and in this respect resemble the cases of "sleep paralysis" in which the patient is aware on awakening of what is happening, but is unable to move.

Whether these attacks are to be regarded as akin to epilepsy depends on our definition, but in some respects they are very close—for example, akinetic epilepsy and cataplexy—as well as in such factors as their periodicity, recurrence, abrupt onset, disturbance of consciousness and short duration. Cases have been reported in which both epileptic and narcoleptic attacks occur.

Much the same may be said of migraine, in which cerebral dysrhythmia is generally absent and movements do not occur, but in which the periodicity, aura and prodromata are often found—as well as rather doubtful statistical evidence of both migraine and epilepsy occurring in the same family or the same individual.

To sum up these remarks, it may be said that many queer turns occur in our patients, and as is so often the case, the more one is interested and looks for them, the more one finds. Many occur in anxiety states and hysteria as well as in organic disorders in the central nervous system. The definition of epilepsy is often difficult, though the electroencephalogram may prove to be of supreme importance. Whether one regards the queer turns as epileptic or not depends to some extent on definition, but also on the clinical picture as a whole. It seems desirable to stress the fact that many queer epileptic turns are rather rare, and that to find them in isolation from other more definitely epileptic manifestations is decidedly uncommon (1% according to Lennox).

In any queer or unusual turns there should therefore be a careful search for other epileptic symptoms before they are regarded as epileptic.

THE DRUG TREATMENT OF EPILEPSY.¹

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THE manifestations of epilepsy are so spectacular and dramatic that attempts to control them by drugs are recorded from the earliest beginnings of medical history. From when Celsus saw epileptics drinking the blood of wounded and dying gladiators, so many herbs, potions and magic elixirs had been acclaimed that Sieveking wrote in 1861: "There is scarcely a substance in the world capable of passing through the gullet of man that has not at one time or another enjoyed the reputation of being anti-epileptic."

When Locock described the value of bromides in 1857, a rational basis for treatment was established at last. Further great advances were made with the introduction of phenobarbitone by Hauptmann in 1912, and with the preparation of phemitone ("Prominal" or "Mebaral") some twenty years later. The introduction of electroencephalography and the rapid progress of neurophysiology during the past twenty years opened new horizons in the understanding of cortical function and enabled the remarkable studies of Merritt and Putnam, which resulted in the discovery of diphenyl-hydantoin ("Dilantin") in 1937. With this, a new era began in the control of epilepsy, and progress has, fortunately, continued with many new and valuable additions to our therapeutic armamentarium.

Today we may be confident of our ability either to inhibit completely or to reduce significantly the number and severity of fits of the vast majority of our epileptic patients. At least twelve different drugs are now available in this country and have proved their worth; a further two have satisfied clinical trials in Great Britain and the United States of America and should reach us within the next few months.

Some of the antiepileptics are sedatives and cerebral depressants, such as the bromides and barbiturates; other more recent ones, the hydantoins, "Phenurone" and "Mysoline", have little or no sedative effect, while the oxazolidine-2,4-dione derivatives, "Tridione" and related compounds, are indeed said to be cerebral stimulants. Therefore we may now discard the old presumption that an anticonvulsant must necessarily be a sedative, and that the patient pays for the control of his fits with drowsiness or a reduction of his working efficiency.

The mechanism of anticonvulsant action is not yet fully understood. This knowledge would presuppose an equal knowledge of the physiology of seizures. At present we know only that Hughlings Jackson's "occasional, sudden, excessive, rapid and local discharge of grey matter" must arise from neurons, whatever the underlying primary cause. It would appear that in some instances these neurons become transiently hyperexcitable, in others they have lost their intrinsic inhibitory mechanisms, and in yet others there may even be a transformation of inhibition into excitation. That inhibition plays an important part cannot be doubted, once we have seen the akinetic attacks so common in children suffering from *petit mal*.

From the therapeutic point of view, Denis Williams's concept of an epileptic brain unduly susceptible to abnormal discharges, and the epileptic process as a separate incident superimposed on such a brain, is most valuable. This means that in the vast majority of cases of epilepsy, certainly in all the genetically determined cases, we have at least two mechanisms to combat: (i) the background of inherent susceptibility and instability of the brain, the constant cerebral dysrhythmia, which determines the nature and degree of spread of the discharge; (ii) the sudden, excessive discharge arising on this background and producing the clinical seizure.

It has been suggested that the sedative anticonvulsants protect the unstable brain from the epileptic discharge, whereas the hydantoins inhibit the epileptic process directly without depressing cerebral activity.

The basic chemical structure of all the clinically useful antiepileptic drugs, excepting the bromides, is similar in spite of the variability in their action against the different forms of epilepsy. Further studies of the mechanism of their action will be of great value for our understanding of the mechanism of seizures.

It was thought until quite recently that any anticonvulsant was equally effective for all forms of epileptic seizures. We know now that a given type of seizure will respond best to one particular drug, and therefore we must "fit the treatment to the fit". With so many effective anticonvulsants at our disposal, the drug treatment of epilepsy has become a fine art, which requires patience, skill and experience on the part of the physician. We must aim at abolishing the fits without producing unpleasant or toxic side effects. This requires a conscientious and painstaking study of each individual patient, of the aetiological factors, type, frequency and time incidence of his fits, and of his reaction to any particular drug.

The drug treatment of epilepsy can begin only after the history and clinical examination, together with ancillary diagnostic investigations, have definitely excluded localized cerebral pathological conditions which may require surgical attention. A careful history, both from the patient and from observers, is most essential, for it will tell us the type of seizures, their frequency, the time of day or night when they occur and their relation to menstruation or emotional upsets, and will often throw light on aetiological factors. On this information we base our choice of drug, of dosage and of the optimal time for administration. For example, it is frequently found in female epileptics that fits occur maximally or even exclusively during the premenstrual or menstrual periods. It would appear that this is causally related to the premenstrual water retention, as hydration is known to predispose to seizures. Therefore we must advise these patients to restrict their fluid and salt intake, give them ammonium chloride and increase the dosage of antiepileptics during these critical days. Again,

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the patient in whom emotional factors appear to precipitate the attacks will require one of the sedative anticonvulsants at least as part of his medication.

An electroencephalogram with expert interpretation is the next essential prerequisite. It will show us the inter-seizure state of the brain and the severity of the dysrhythmia, and will often determine the type of seizure. At times, but not often, the electroencephalogram disagrees with our clinical impression, and then we have to weigh the reliability of our history against the limitations and fallacies of the electrical record. Our final decision will necessarily have to vary with each individual case.

When we have determined the type of seizure, *grand mal*, *petit mal* or psychomotor attacks, we can decide on the drug most likely to be effective. The dosage will vary with the frequency of seizures, the degree of abnormality of the electroencephalogram, and the patient's individual reaction to the drug. When the epilepsy is post-traumatic or secondary to cerebral damage, a higher dose is usually needed than in "idiopathic" or genetic cases. If the fits are mainly nocturnal the maximum dose must be given before retiring; if they are diurnal, the drugs should be spaced equally throughout the day. Children generally tolerate the anticonvulsants well, and one frequently finds that failure to control their fits is due to inadequate dosage, though it may have been correctly calculated for their age and weight.

Grand Mal.

For the control of *grand mal* or major convulsions some eight drugs are now available in this country; phenytoin sodium, methoin, phemitone, phenobarbitone, "Rutonal", "Superminal", bromides and "Phenurone".

Phenytoin Sodium.

Phenytoin sodium (diphenylhydantoin), also known as "Dilantin" or "Epanutin", is effective against *grand mal* in some 60% of cases. In a small percentage of cases it may control psychomotor seizures, but is ineffective against *petit mal*, which indeed it may aggravate. It is a better anticonvulsant than barbiturates and does not produce drowsiness. The average dose for adults is 0.3 gramme (4.5 grains) daily, and this may be gradually increased up to 0.6 gramme (9.0 grains), though at this level many patients will begin to show signs of intolerance. Phenytoin should be taken with meals, as it is strongly alkaline and may produce gastric distress, nausea and vomiting during the early weeks of treatment. The commonest side effect is hyperplasia of the gums, said to occur in about 30% of cases. It causes little discomfort and does not usually require discontinuation of the drug. In many cases it diminishes with good oral hygiene and a diet high in vitamin C content. Rarer toxic manifestations are dizziness, ataxia, blurring of vision, diplopia and tremors. These usually appear during the first ten days of treatment and clear up spontaneously in spite of continuation of therapy. In less than 5% of cases erythema or macular and purpuric rashes may occur, and an occasional case of exfoliative dermatitis has been reported. Suspension of treatment is then necessary and almost invariably results in complete disappearance of the skin lesion.

Methoin.

Methoin (3-methyl-5-phenyl-5-ethyl-hydantoin), or "Mesantoin", first introduced in 1945, is effective mainly against *grand mal*, and occasionally in psychomotor seizures if these are associated with *grand mal*. It is stated to be particularly successful in the control of Jacksonian attacks, and some reports claim abolition or significant reduction of seizures in up to 70% of cases. Treatment with methoin must be introduced gradually in order to avoid undesirable side effects. It is marketed in tablets of 0.1 gramme. Initial dosage should be one-half tablet daily for the first week, with gradual increments of half to one tablet daily at weekly intervals until a maintenance dose is reached of four to six tablets per day (0.4 to 0.6 gramme). In severe or resistant cases this may be carefully increased at fortnightly or even monthly intervals

up to ten tablets (1.0 gramme) per day. Drowsiness is the most common side effect but can usually be relieved by the administration of "Dexedrine". Ataxia is not uncommon and some 5% of patients develop morbilliform or urticarial rashes. The most serious danger, though it is rare, is a depression of granulocytes. We must therefore advise patients taking methoin to report any sore throats immediately, and periodic leucocyte counts are advisable with cessation of administration of the drug once the number of granulocytes falls below 1600 per cubic millimetre. As "Tridione" may also cause a granulopenia, the combination of this drug with methoin is not advised for patients suffering from both major and minor seizures.

During the past five years several other hydantoin derivatives have been reported as clinically useful in the American literature, but usually they have been found to be inferior to phenytoin or methoin.

Phemitone.

Phemitone (3-methyl-5-phenyl-5-ethyl barbituric acid), known also as "Prominal" or "Mebaral", is a barbiturate, but does not cause quite the same degree of drowsiness as phenobarbitone. It is of most use in the control of *grand mal*, though deteriorated epileptics and those whose condition is secondary to brain damage do not usually respond well. Three grains of phemitone are roughly equivalent to 1.5 grains of phenobarbitone, or 15 grains of potassium bromide. The dose varies from three to nine grains daily, and two grains twice daily suffice for most patients if given in conjunction with phenytoin. There are no serious or unpleasant side effects except lethargy, which can be alleviated by "Dexedrine".

Phenobarbitone.

Phenobarbitone has stood the test as an efficient anticonvulsant for some forty years. Its main value is in the control of *grand mal*, whether genetic or post-traumatic, and it seems to benefit particularly patients with severe and frequent fits. Mildly affected patients with infrequent seizures and old patients with fits due to cerebral vascular degeneration do not respond so well. Phenobarbitone is of no use in *petit mal*, except for occasional patients with predominantly myoclonic attacks. Up to five grains daily may be prescribed, but the main drawback is drowsiness and occasionally depression, apathy and interference with working capacity. Rarer toxic reactions include ataxia, slowing of speech, asthenia and irritability, and very rarely dermatitis. Although children generally tolerate phenobarbitone well, it may at times produce intense irritability and excitement and must then be abandoned. I have seen one child, four years old, whose speech deteriorated severely while he was taking phenobarbitone, and improved rapidly when the drug was stopped.

If phenobarbitone is stopped abruptly a dangerous increase in the frequency and severity of fits will occur in over 70% of patients, and *status epilepticus* is prone to develop. Therefore, when a patient is changed from this drug to another anticonvulsant, it is most essential that this be done cautiously and gradually. This feature of phenobarbitone is also a disadvantage with unreliable patients, who may forget to take their tablets or allow themselves to run out of a supply.

"Rutonal" and "Superminal".

"Rutonal" and "Superminal" are other proprietary barbiturate derivatives which may be helpful in some selected cases when other drugs have failed.

Bromides.

Bromides still have a place in the treatment of major epilepsy, but have many unpleasant side effects and should be used only when other drugs have proved ineffective or toxic. Sodium bromide is probably the preparation of choice, or a mixture of three bromides may be used. Beginning with 45 grains per day, the dose may be gradually increased up to 90 grains. A decreased, but constant, chloride content of the diet causes more of the bromide to be retained and permits of lower dosage. The addition of three minims of *liquor arsenicalis* to the mixture often

prevents the development of acne, but if continued for long periods this may lead to arsenical intoxication. As the excretion of bromides is very slow, an occasional missed dose does not matter, but a constant watch for signs of intoxication is essential.

"Phenurone" and "Mysoline".

Two new and effective anticonvulsants, "Phenurone" and "Mysoline", have been reported from the United States of America and England, but are not yet freely available in this country.

"Phenurone" (phenacetylurea) was found successful in some 50% of patients resistant to other drugs and is claimed to be particularly valuable in the control of psychomotor seizures. Toxic effects include skin rashes, granulopenia, gastro-intestinal disturbances and occasionally severe liver damage, which has resulted in death in a few cases. As some 20% of patients also develop unpleasant personality and psychological manifestations, the drug was wisely withheld from the market for some three years and will have to be used with great caution when it becomes freely available.

"Mysoline" (5-phenyl-5-ethyl-hexahydropyrimidine-4:6-dione) was reviewed in *The Lancet* last year and appears to be most useful in the management of major convulsions with relatively few side effects. A controlled clinical trial of "Mysoline" in this country is now in progress, and present indications are that it promises to be a most valuable drug. No serious toxic effects were encountered, but a considerable proportion of patients complain of drowsiness, lethargy, ataxia and disturbances of visual accommodation, which usually wear off after a few weeks in spite of continued administration. It is likely that "Mysoline" will be freely available in Australia later this year, and it is hoped that further reports about it will appear in the literature before then.

General.

At present I would suggest that the treatment of patients with major seizures should begin with a combination of phenytoin and phemitone. The patient should be asked to keep an accurate diary of his fits, and the success of treatment and occurrence of side effects is reviewed during later visits. The dosage can then be adjusted or redistributed, or one or other drug gradually changed to a different anticonvulsant, until the combination most effective for each individual patient is found.

Petit Mal.

Three drugs, troxidone, "Paradione" and "Malidone", are at our disposal for the control of the *petit mal* triad, which includes pyknolepsy, akinetic attacks and myoclonic jerks. Another, "Milontin", was found useful overseas, but is not yet freely available here.

Troxidone.

Troxidone (3,5,5-trimethyl-oxazolidine-2,4-dione) or "Tridione", introduced by Richards and Everett in 1944, effectively controls or reduces the frequency of any of the three components of the *petit mal* triad in about two-thirds of cases. It does not control *grand mal*, but indeed may make it worse. An effective dose for adults is 0.3 gramme three times a day, and this may be cautiously increased up to 0.6 gramme three times a day. Unfortunately toxic effects are not infrequent. Photophobia or an impression as if objects are covered in snow occurs in about 22% of cases, but often clears up after a few weeks of treatment. Patients can relieve this phenomenon by wearing dark glasses. Various skin eruptions, including exfoliative dermatitis, occur occasionally and call for withdrawal of the drug. The real danger lies in depression of bone marrow activity, said to occur in about 7% of cases. This usually manifests itself first as granulopenia, but may progress to fulminating and fatal pancytopenia. It is therefore essential to keep the patient under constant supervision with regular blood counts and a careful watch for any throat infection. If the absolute neutrophile cell count falls below 2500 per cubic millimetre more frequent checks are indicated, and

if it falls to 1600 per cubic millimetre the drug must be withheld.

"Paradione."

"Paradione" (3,5-dimethyl-5-ethyl oxazolidine-2,4-dione) occasionally succeeds when "Tridione" fails, but has the same toxic effects and requires the same precautions. Dosage is the same as that of "Tridione".

"Malidone."

"Malidone" (3-allyl-5-methyl-oxazolidine-2,4-dione) is reported to be as effective as "Tridione" without producing photophobia. The danger of marrow depression, however, remains, and dizziness appears to be another occasional side effect. It is given in a dose of 0.3 gramme three times a day, which may be cautiously doubled in resistant cases. "Malidone" has only recently become available in this country, and further evaluation by careful clinical trials will be helpful.

"Milontin."

"Milontin" (methyl-phenyl-succinimide) is reported to be as useful as "Tridione", but produces disagreeable side effects in about 22% of cases. It is not yet freely available here.

General.

It is my practice to begin the treatment of patients suffering from *petit mal* with "Tridione", and if this does not reduce their attacks sufficiently, to try them successively on "Paradione" and "Malidone". With some patients who respond only partially to "Tridione", "Paradione" or "Malidone", the addition of "Dexedrine", or an attempt at a ketogenic diet, may bring about a further reduction in the number of their fits.

Psychomotor Seizures.

The control of psychomotor seizures, the episodes of bizarre motor activity, sensory hallucinations or illusions, unprovoked temper outbursts, fugues and automatisms, is still the bugbear of epileptic therapy. Almost any new antiepileptic is claimed by the manufacturers to be effective against these attacks, but usually proves disappointing with further clinical trial. At present only "Phenurone" appears to control this type of seizure and "Mysoline" may hold a promise, but phenytoin and methoin help occasionally, particularly in patients who have also major convulsions.

In patients with more than one variety of seizure, two or more specific drugs have to be combined. Phenytoin and troxidone provide often the most effective combination when both *grand mal* and *petit mal* occur.

Status Epilepticus.

Status epilepticus is a medical emergency and may result in the death of the patient if adequate treatment is not immediately instituted. The injection of eight to ten millilitres of paraldehyde into the gluteal muscle is the method of choice and succeeds in the majority of cases. If the fits continue, five millilitres of paraldehyde given intramuscularly may be repeated at half-hourly intervals until they stop. In desperate cases, six to eight millilitres of sterile paraldehyde may be given by slow intravenous injection, or intravenous injection of thiopentone may be used in a dose of 0.25 gramme for an adult, followed by intramuscular injection of paraldehyde if necessary. Alternatively, three to five grains of sodium phenobarbitone may be injected subcutaneously or intramuscularly, or the patient may be anaesthetized with "Avertin" or ether. The rectal administration of chloral hydrate helps in some of the milder cases.

Conclusion.

There is no doubt that we can now secure relief for the majority of our epileptic patients with an intelligent and conscientious use of drugs. It must be our object to secure abolition of fits for a sufficient length of time to enable the patient to lose the epileptic habit, and treatment must be continued for at least three years after the attacks cease; it may then be abandoned gradually.

Much remains to be learned about the origin of seizures and the mechanism of action of anticonvulsants, but we

may be confident that even better chemical therapy will be developed in the near future.

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PSYCHOLOGICAL AND SOCIAL MANAGEMENT OF THE EPILEPTIC PATIENT.¹

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PSYCHOLOGICAL and social aspects of chronic illness always need consideration if the best results are to be obtained by treatment. The disorder we are discussing today exemplifies this particularly well. Epilepsy is not a specific disease. In healthy persons the universal susceptibility to convulsions is manifest only under extreme provocation, whereas in the epileptic the sudden and recurrent lapses of consciousness are brought on by lesser and perhaps different stimuli. Moreover, the interval between attacks may vary from minutes to years. It is thus preferable to talk of the epileptic patient rather than of epilepsy, and to regard those afflicted as suffering from a symptom complex which has a wide variety of causes, manifestations and effects. Whilst accurate diagnosis and skilful use of drugs are of major importance in treatment, each patient also presents a personal and a public problem. This psychological and social aspect is not only significant in the management of the illness, but at times may influence its causation. We must remember to treat the patient and not the "disease".

Psychological Management.

When the diagnosis of epilepsy is established—not always an easy matter—I believe that the patient or, in the case of children, the parents should be informed. It is seldom, if ever, wise to hedge or to fall back on such euphemisms as "it's only convulsions". As the interview at which this diagnosis is explained and discussed is likely to be crucial, ample time should be set aside for this purpose. At once the doctor is likely to be confronted with a number of fears and anxieties, some real, some exaggerated, some imaginary. Foremost amongst these seems to be the fear that as epilepsy is a disorder of the brain it will of necessity cause progressive mental deterioration, if not madness. This attitude is in part a carry-over from the "dark ages" from which we have not yet fully emerged, when mental disorder was regarded as a demoniac possession, and in part due to the fact that in a small minority of cases the brain abnormality responsible for the epilepsy also causes some deterioration. This is most often seen after cerebral birth injury, and less frequently with cerebral arteriopathy. Also, when bromide was the only anticonvulsant drug available, overdosage with resultant bromism led to dementia. It should be pointed out that epilepsy does not, of itself, cause mental deterioration. The next popular fear is that little or nothing can be done in the way of treatment. Some may think it less justifiable to be reassuring on this point, because in perhaps 25% of cases this turns out to be more or less true; but I believe it is most important to stress the improved and improving results of modern investigation and treatment. This is desirable, not only on humanitarian grounds, but also to aid the patient's morale—an important factor both in treating the fits and in helping the patient to face the difficulties inherent in the life of an epileptic. It should be made clear that treatment must be continuous and prolonged, and that the epileptic tendency may persist. In this respect a parallel may be drawn with some more "respectable" condition, such as diabetes. Patients or parents with average intelligence and stability understand and are helped by such justified optimism; but those with obviously neurotic personalities may unconsciously use their illness in their flight from reality. If so, their neurosis will need additional psychotherapy directed to alleviating the underlying personality disorder. Not only does the right doctor-patient relationship inspire confidence through better understanding of the problem, but the patient is encouraged by feeling that

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his doctor is taking some responsibility for his progress, is prepared to accept and sympathize with him, and is readily available to discuss and advise on difficulties as they may arise. In this role the doctor is not unrewarded. Occasionally he effects a cure, often an improvement. When this is not possible he can always comfort and encourage.

Many other psychological difficulties will be encountered, varying from patient to patient. The recurrence of fits, often at unexpected and embarrassing times—particularly if no warning occurs—tends to destroy confidence and inhibits freedom of action. Then in some cases there is fear of injury or even of death. With regard to the former, it is a fortunate dispensation that serious injury seldom occurs. Lennox and Cobb have in fact stated that the accident-proneness of the epileptic is no higher than that of other workers. Occasionally patients with major seizures fall in such an attitude as to inflict a recurring injury.

For example, I recently examined a young man who always falls forward with both arms outstretched. This ultimately led to recurrent dislocation of the right shoulder necessitating operation.

The fear of a recurrence of this injury is a real anxiety. Sometimes the aura itself is unpleasant or even terrifying.

A little boy, aged three and a half years, in his aura feels that everything is going round and going to fall on top of him. If he is outside, he runs in; if he is inside, he wants to get out, and always screams for his mother.

Interestingly enough, the recollection of this experience has caused this child to be upset by the sight of anything going round and round. He is too young for explanation, and all one can advise is constant reassurance, combined with a minimum of fuss—not an easy feat for a worried mother. As a sidelight on human nature, I may mention that his little brother, only one and a half years old, has observed this fear and delights in running round and round in circles to make his brother scream. Such an episode tends rather to shake one's belief in the essential goodness of human nature, and I sometimes feel that the "original sin" school may be nearer the truth.

Fear of death in a fit has little foundation in fact, and patients can be unreservedly reassured. Any risk is associated with occupational hazards, and even here is a very rare occurrence. If after reassurance death fears persist, it is likely to be based on some other associated mental disturbance. With children especially, but also with adults, feelings of rejection and resentment are not infrequent, based on the attitude of parents and of members of the community, and perhaps increased by restrictions and deprivations that may be a necessary part of treatment. With young children this must be handled through the parents and will vary in detail from family to family. In general, parents should be advised to treat the child as naturally and normally as possible, without excessive sympathy and anxiety on the one hand or disappointment or differentiation on the other. The fit and its aftermath should always be treated in the most matter-of-fact manner, without fuss or comment. When anxiety in the parent is based on guilt feelings, it may be harder to handle, arising as it often does from some deep conflict—at times associated with the epilepsy, as when parents with an epileptic family history feel they were wrong to have had children, at times due to other emotional disturbances.

If in spite of treatment severe fits continue to be fairly frequent, it is not surprising that some degree of hopelessness and despair should develop. It is in these cases—fortunately a minority—that the family, social and occupational set-up is of such significance. The aim is to provide, through these agencies, the maximum degree of compensation and emotional satisfaction. But here again, as in almost everything concerning epilepsy, one should not generalize. Not infrequently one comes across chronic epileptics who manage to achieve and preserve an amazing degree of cheerfulness, in spite of, or perhaps because they have adjusted themselves to, their disability. I mentioned a moment ago the question of heredity. Almost all epileptics who wish to marry, or are already married, are anxious about whether they should have children. Because

much remains to be learnt concerning the role of heredity in the production of epilepsy, one again cannot be dogmatic. On present evidence it seems that hereditary factors play an important—some would say all-important—part in the underlying predisposition. But those "trigger" mechanisms that activate the predisposition may be, and sometimes undoubtedly are, of major aetiological significance. When one adds to this the fact that the inherent tendency may be only latent and that it is often impossible to obtain an accurate family history, it will be clear that in any one instance accurate prognosis is seldom possible. All one can then do is to refer to general probabilities, and fortunately these are relatively reassuring. It is at present widely believed that when one parent is epileptic the chances of epileptic offspring are about 1 in 40. When neither parent is a known epileptic the chances are not so very much less—say 1 in 200. Factors warranting a more favourable prognosis would be evidence that brain injury—accident or infection—preceded the epilepsy or that the onset of seizures was later than childhood. Perhaps the most important factor would be for the other parent to be free of any predisposition to seizures. With a history of epilepsy in both parents, or very strongly in the family, the chances would be appreciably greater, and it is members of this group only, I believe, who should sometimes be advised against having children. Unfortunately some of these neither seek nor accept advice of this sort; I refer to a small number (about 10%) who are mentally defective. For some of these sterilization should be considered, not only because of the risk of epileptic children, but because the parents may be incapable of looking after a family.

Whilst personality disturbances of these and other varieties often arise out of the epileptic life situation, sometimes psychological factors may enter into the causation of fits, acting as one of the trigger mechanisms. In some of the literature on the subject—as, for example, Bridge's book—this process is regarded as frequently occurring, and great attention is paid to it. It is stated that treatment of emotional disorders will of itself lead to a diminution in the number of fits. However, as such patients are usually under drug therapy also, and as much remains to be learnt about the mechanism of seizures, it is often difficult to come to a definite opinion. I seem to have noticed myself that if one takes a psychiatric history from many patients with whatever illness one will find evidence of real conflicts and emotional disturbances. However, such a finding does not prove that they are or are not causative factors in any particular instance. Then in most of my epileptic patients the fits seem unrelated to any particular set of factors and are unpredictable. Only occasionally have I felt satisfied that emotional stresses have produced either the first or subsequent attacks. I should be most interested to hear the opinions of other speakers on this question. It is important to realize that the peculiarities of personality and character sometimes found in the epileptic child and adult are not unique, but similar to those found in persons with other handicaps and deprivations, and that they will similarly be modified by the make-up of the patient and the environment at home and in society. The concept of the "epileptic personality" as inevitably morose, aggressive and disgruntled has no foundation in fact, and this statement is not negated by the admission that some epileptics are more irritable and difficult soon after their fits or if they are having fits frequently.

Lastly under this heading, mention should be made of epileptic psychotics. Like the major psychoses, the aetiology of epileptic psychosis is obscure. It is relatively uncommon, and the proportion of patients in the community remains fairly constant. These patients are paranoid in type and often very suspicious, aggressive and difficult to handle. Sometimes their psychosis undergoes remission, either spontaneously or with shock treatment, when they can be released on trial leave; but it tends to recur, and often they are safer kept permanently in mental institutions.

Social Management.

At the same time as medical treatment is being carried on the social adjustment of the patient has to be attempted. Whilst some of the difficulties encountered are inevitable so long as the fits continue, much of the unhappiness and frustration of the epileptic is due to misconceptions and lack of reasonable cooperation on the part of others in the community. Few people give much thought to the problem or are aware of its magnitude. Whilst it is rare to see a person in an epileptic seizure, it is estimated that one in every three to four hundred persons is an epileptic. Some even make the figure as high as one in two hundred. The disorder is thus as frequent as tuberculosis or diabetes. At the Johns Hopkins Hospital, where Bridge conducted his clinic, one child out of every 60 to 70 had epilepsy, and one in every 20 to 30 had had some convulsive disorder, this latter higher figure being due to conditions not epileptic, such as tetany, hypoglycaemia, lead poisoning, hysteria, brain tumour, subdural haematoma and febrile convulsions. Such figures far exceed the incidence of rheumatic fever, asthma or tuberculosis in children. Whilst facilities for the prevention and treatment of these diseases are becoming adequate, the care of epileptics is in the main inadequate and non-coordinated, and with a few exceptions seems no one's particular business.

What are the causes of this relative indifference, and can they be overcome? In the forefront is the natural repugnance, if not actual fear, that most people feel at the sight of a major fit. Coupled with this is the attitude of mind, still too prevalent, that regards epilepsy as a form of mental disease and as such something to be ashamed of and avoided. In the United States of America for some years there have been two organizations interested in the care of the epileptic. These have recently united to form the National Epilepsy League, with headquarters at 130 North Wells Street, Chicago. Through the kindness of the American Psychiatric Association I have received some of their literature written for the guidance of the epileptic patient and for the education of the public. With the motto "Facts, not Superstition" it seems that they are doing work of considerable value. Their pamphlets include such titles as "The Employment of Epileptics", "Marriage and Children for Epileptics", "Epilepsy a Problem in Public Health", "What's Going to Happen to Me?", "Prognosis, Favourable" and "Epilepsy, the Ghost is Out of the Closet". In addition to factual information there runs through them a note of optimism and hope, and the emphasis throughout is to treat the epileptic as a normal human being. If at times this attitude may be a little exaggerated, it is, I think, under all the circumstances justifiable.

Suitable occupation is of paramount importance, not only as an aid to morale, but because it tends to decrease the frequency of fits. The 60% to 70% of epileptics whose fits under modern methods of treatment are relatively infrequent and who are otherwise mentally and physically healthy, have only to avoid occupations in which they or other persons might be injured if a fit occurred. This excludes working at heights, near moving machines or fires, or taking jobs involving the safety of others. The trained social worker will often be needed as a link between the patient, the industrial field and the community. When one remembers that the handicapping effect of epilepsy varies from nearly 100% to nearly zero, it is clear that choice of occupation is an individual problem. As well as the need to consider the nature, severity and frequency of attacks, certain mitigating features may be present, such as the occurrence of fits only during sleep, or the presence of sufficient warning to enable the patient to sit down, which is stated to occur in about half of all patients. Lennox and Cobb in an article on the employment of epileptics state the following conclusion:

A certain proportion of adult Epileptics because of brain injury which has caused paralysis or mental impairment, peculiarities of personality or the frequency of severe seizures are not fit for employment. This proportion is small—probably no more than a quarter of the patients in the community.

For many of these—in England more particularly—specialized workshops have been set up.

As in about 75% of cases epilepsy begins before the age of twenty years, and in most of these it begins in childhood, the question of schooling is a major problem. As a general rule, unless the fits are fairly frequent—occurring, for example, more than once every week or two—or unless mental retardation sufficient to interfere with schooling is present, every effort should be made to have the child attend an ordinary school. The cooperation of the teacher will usually be obtained if the right approach is made. If a fit occurs in class, the others should be told the child is sick and the patient allowed to lie down till he has recovered and then readmitted. The occurrence should be treated in the most matter-of-fact way with a minimum of fuss. If the fits are of *petit mal* variety, often no notice need be taken of them at all. When fits are very frequent in spite of treatment, or when retardation is present, admission to a residential institution may be desirable.

In Australia I think we have only one, the Talbot Colony at Clayton, opened in 1907. This has facilities for 100 persons, and the Victorian Education Department runs a special school for children from the age of five to sixteen years. In 1951, 14 children were enrolled, though more could be admitted. Adults suitable for such care are also those more or less mentally, physically or socially incapacitated. Some remain for long periods and on an average about 30 are discharged each year. Another important need in the social adjustment of the epileptic is better education of the public concerning this disorder. If superstition could be replaced by fact, many of the difficulties and much of the unhappiness at present obtaining would disappear. As has been apparent from the papers presented in this symposium, epilepsy is so extensive, so complex and so important that no one person has the expert knowledge or the time to deal with patients single-handed: there must be team-work. If this is forthcoming, not only will the ghost be out of the closet, but some day it may even be laid.

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A MICROTECHNIQUE FOR MEASURING THE HÆMATOCRIT VALUE.

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MOST methods for determining the hæmatocrit value—the percentage volume concentration of red blood cells in a sample of blood—are based on the same principle. A sample is centrifuged in a glass tube of uniform bore, and the length of the column of tightly packed cells is expressed as a percentage of the total length of the column of blood. Many different types of hæmatocrit tubes have been used since Hedin first introduced the test some sixty years ago, but that used in most laboratories at the present time was devised by Wintrobe (1933). Wintrobe's method is unquestionably satisfactory for most hæmatological purposes, but requires at least one millilitre of blood. There are occasions when it is difficult to obtain this amount, especially from infants and children and from small laboratory animals. Various microtechniques requiring only small quantities of blood have been described by Miller (1939), by Hamre (1940), by Guest (1938) and by Meyerstein (1942). However, these methods, with the exception of Meyerstein's, all require special precautions in sealing the tubes to prevent leakage of blood.

A further microtechnique has been developed in an attempt to overcome this and some other disadvantages of earlier methods and is described in this paper.

¹This work was undertaken during tenure of a Fellowship of the National Health and Medical Research Council.

Methods.

Capillary bore glass tubes 90 millimetres long, of 0.8 to 1.0 millimetre outside diameter and 0.4 to 0.6 millimetre inside diameters, are used. They are labelled with appropriate coloured pencil markings. A small gas jet for sealing the capillary tubes is constructed from a short intradermal needle, and the flame is protected by a piece of glass tubing (Figure 1). Heparin (Evans) in the concentration of ten international units per millilitre of blood is employed as the anticoagulant. The tubes are filled with suitably diluted heparin solution, which is dried in a glass desiccator with silica gel or calcium chloride. Non-heparinized tubes were used with heparinized venous blood for some of the experiments. The filled hæmatocrit tubes were centrifuged at 3500 revolutions per minute with a mean radius of rotation of 12.5 centimetres. It was found that maximum packing of red cells occurred in

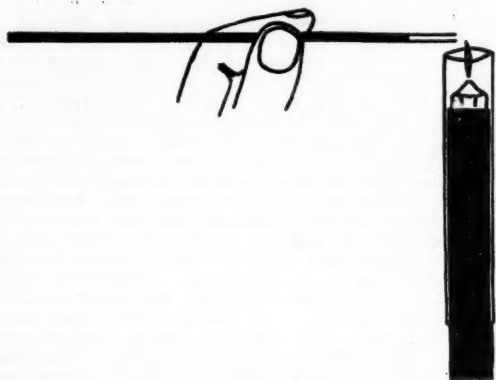


FIGURE 1.
Method of sealing microtube.

fifteen minutes in the microtubes and in thirty minutes in the Wintrobe tubes. The lengths of the columns of blood in the microtubes are measured in a shallow groove one millimetre wide, cut in a small piece of "Perspex". A strip of metric ruled paper is pasted to the "Perspex" on one side of the groove.

The expression "capillary blood" is used for blood obtained when the finger is pricked with a needle.

Technique of Test.

The tubes are filled with blood by capillary attraction to give a column 65 to 75 millimetres long. The end of the tube which does not contain blood is then sealed in the flame, care being taken to avoid overheating of the blood (see Figure 1). During the sealing the column of blood should not move. A number of sealed tubes are placed in a flat-bottomed test tube which fits the bucket of the centrifuge and packed with cotton wool. They are centrifuged for fifteen minutes and measurements of the columns are recorded. The free ends of the tubes are now sealed and centrifuged for a further fifteen minutes. A second reading is then made and the hæmatocrit value calculated from the two sets of readings. Duplicate determinations in the same tube reduce errors due to irregularity in the bore of the tubes. The use of precision bore tubing would eliminate the necessity for duplicate estimation.

Experimental Observations.

To provide an estimate of the error of replicate determinations, eight microtubes were filled with heparinized blood from the same sample. The hæmatocrit value was determined in each tube by the technique of duplicate measurements described above. The eight values so obtained are shown in Table I. The mean and the standard deviation of the values from the mean are 49.37 ± 0.27 and 0.78 ± 0.19 respectively.

Venous blood obtained from 14 volunteers was mixed with heparin in test tubes. The hæmatocrit value of each sample was determined in both microtubes and Wintrobe tubes. Capillary blood was also drawn from 10 of the subjects into heparinized microtubes and the hæmatocrit values were determined. The results are shown in Table II.

The mean difference between hæmatocrit values in microtubes and those in Wintrobe tubes on venous blood is

TABLE I.
Replicate Measurements of the Hæmatocrit Value in Microtubes.

Microtubes.	Hæmatocrit Value.
1	49.9
2	48.6
3	49.8
4	49.1
5	49.0
6	50.3
7	49.1
8	49.2
Mean	49.37 ± 0.27
Standard deviation	0.78 ± 0.19

0.21 ± 1.28 . The individual differences range between -1.3 and $+1.1$. The difference between the mean of the hæmatocrit determined by Wintrobe's method in venous blood and the mean of values obtained on capillary blood is 0.82 ± 1.85 . The individual values vary between -4.2 and $+2.7$. Neither the difference between the mean values obtained in the two types of tubes nor the difference between the mean value of venous blood and that of capillary blood is statistically significant.

TABLE II.
Comparison of Hæmatocrit Values on Venous and Capillary Blood.

Number of Subject.	Venous Blood.			Capillary Blood, Microtubes.
	Microtubes.	Wintrobe Tubes.		
		I.	II.	
1	39.3	37.0	37.0	34.3
2	39.1	39.0	39.0	37.8
3	38.4	39.0	39.0	41.6
4	40.5	40.8	40.8	40.1
5	40.9	41.6	—	—
6	42.5	42.8	—	—
7	41.7	42.8	42.8	47.0
8	42.5	43.5	43.5	45.7
9	44.3	43.9	—	—
10	43.9	44.6	44.6	42.3
11	46.2	45.8	45.8	49.9
12	46.4	46.0	46.0	45.1
13	47.1	47.4	47.4	50.3
14	48.5	49.0	—	—
Mean	42.88 ± 0.80	43.00 ± 0.91	42.59 ± 1.11	43.41 ± 1.65
Standard deviation	3.31 ± 0.63	3.4 ± 0.68	3.5 ± 0.78	5.2 ± 1.16

Discussion.

No claims are made that this technique is superior to other microtechniques. It must be pointed out that other methods, with the exception of Meyerstein's, rely for the sealing of the tubes on sealing compounds, rubber bands, adhesive tape or mechanical clamping devices. Not only do these increase the amount of apparatus required, but none is entirely satisfactory, because leakage of blood not infrequently occurs during centrifuging. This has been mentioned by one of the authors quoted (Hamre, 1940) and also by Wintrobe (1946) when referring to Guest's technique. Wintrobe developed his permanently sealed tube to overcome the difficulty of sealing. Meyerstein's

tube is also permanently sealed at the bottom, but as the top is cup-shaped only one tube can be placed in a centrifuge bucket.

The advantages of the expendable microtube described in this paper are its small capacity, its small over-all size and the safe method of sealing. The error of measurement is greater than the error of the Wintrobe method (0.59%), but comparable with that of other micromethods. The values obtained in the microtubes do not differ significantly from those obtained in Wintrobe tubes (Table II).

The differences between the hæmatocrit values of venous blood (Wintrobe's method) and those of capillary blood (microtube technique) of the same subject are due, in part at least, to differences in the cellular concentration of blood from the different sources. A similar variability of hæmoglobin values determined simultaneously on venous and capillary blood was observed by Walsh *et alii* (1952) and was thought to be related to the condition of the capillary bed at the site of collection.

After this microtechnique had been developed attention was drawn to a similar method described by Parpart and Ballantine (1943).

Summary.

A technique is described for measurement of the hæmatocrit value of blood samples of less than 20 cubic millimetres. It combines the advantages of small over-all size with a safe method of sealing the tubes. The error of measurement was determined and an experimental comparison was made with the standard Wintrobe technique.

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MURRAY VALLEY ENCEPHALITIS: THE CONTRASTING EPIDEMIOLOGICAL PICTURE IN 1951 AND 1952.¹

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THE outbreak of Murray Valley encephalitis in 1951 has been recorded in a series of papers (Special Article, 1951; Anderson, 1952; French, 1952; Robertson and McLorinan, 1952; Robertson, 1952; Anderson *et alii*, 1952). The first section of this paper is largely a summary of portions of those reports; the second section describes briefly the epidemiological surveys conducted during the summer of 1951-1952.

The Situation in 1951.

Murray Valley encephalitis (MVE) was found in clinical form as far east as Albury, and in the west was most concentrated in and around Mildura. Miles, Fowler and Howes (1951) have described the appearance of the disease in the Murray Valley in South Australia.

The 41 patients studied in the outbreak developed their illnesses during the first three and a half months of 1951. The wide dispersion of these cases in both time and space immediately suggested either that the causal virus produced a large number of subclinical infections in humans, or that the virus was a common parasite of domestic or native fauna. Eventually, both these possibilities were proven to be correct.

Soon after the isolation of the virus in chick embryos a wide survey was made of so-called normal human sera from cities and towns scattered throughout the three States of eastern Australia, and from New Guinea.

In and near Mildura, for example, 573 individuals gave sera, which were examined by a complement-fixation test, to estimate antibody to Murray Valley encephalitis virus. The result indicated that in the far north-west of Victoria 20% of adults and 7% of children under fourteen years of age had been infected with the virus of Murray Valley encephalitis.

Similarly, complement-fixing antibody was detected in sera sent to Melbourne from Mornington Island in the Gulf of Carpentaria, and also in sera from various towns in western New South Wales and in the eastern coastal belt of Queensland and from Bulolo in New Guinea. It was thought that this complement-fixing antibody could be accepted as evidence that the virus had been active in those areas in the summer of 1951; although, particularly in Queensland, there was little evidence that it had caused severe human encephalitis during 1951. The reason for some doubt in this interpretation was the possible interference with the Murray Valley encephalitis test by antibody to dengue and perhaps other viruses. In order to reduce this source of confusion, a proportion of the above positive sera from northern Australia has been examined more recently in an intraperitoneal baby mouse neutralization test. The positive results obtained confirmed the belief that the virus had produced subclinical human infection in western New South Wales, Queensland, Mornington Island and New Guinea.

Attention then turned to wild and domestic birds in the Murray Valley, most of this work being centred in Mildura. In that area, 99 adult water-birds were taken during the late winter and spring of 1951, and 40 were shown to carry serological evidence of previous infection with the virus of Murray Valley encephalitis. These "positive" birds included 12 species. Again, of 60 land birds examined 11 carried antibody to Murray Valley encephalitis. The infection had indeed been heavy in the native birds in 1950-1951 (Anderson, 1953).

Meanwhile in the laboratory it had been shown that infection of birds leads to viraemia. The theory was therefore advanced that Murray Valley encephalitis is primarily an infection of wild birds, and that the virus is transferred from bird to bird by one or more types of biting insect. The most probable vector is believed to be a mosquito; on those occasions when virus is abundant in the bird population, and when suitable types of mosquitoes are available to transfer the virus to man and other animals, this transfer takes place. The result is widespread subclinical infection of humans and the advent of a small proportion of severe and even fatal cases of encephalitis.

This concept focused attention on mosquitoes as the vectors of avian, animal and human infections, and evidence was sought regarding the type or types of mosquito capable of acting as vector. A finding that was of considerable interest in this regard was the distribution of neutralizing antibody in domestic fowl in the Mildura area in the winter of 1951. Flocks of chickens normally spend their whole life in one limited area; so that the occurrence of antibody in a flock means that the virus has been introduced into that fowlyard by the vector.

In those Mildura flocks living near the Murray River and exposed to river-bottom mosquitoes, there was a high proportion of positive sera (22 of 31 tested). In flocks some miles distant from the river, and also in flocks living in city backyards in Mildura, there were few positive sera (three of 25 tested). This suggests strongly that mosquitoes

¹ A shortened form of this paper was presented at the Eighth Session of the Australasian Medical Congress (British Medical Association), in August, 1952.

breeding in and near the river are the predominant vectors for the infection of domestic fowl, and probably of man.

Several species of mosquito might answer this description, but in view of the geographical distribution of the infection, and by analogy with the arthropod-borne encephalitis of Japan and America, the mosquito *Culex annulirostris* might be first suspect as the major vector.

In summary, there were 40 known severe cases of Murray Valley encephalitis in Victoria and southern New South Wales in the first three and a half months of 1951. There was a widespread subclinical infection of humans. There was a widespread infection of wild birds, particularly water-birds, and of domestic fowls living near the Murray River. Horses, foxes and opossums also shared heavily in the infection.

The Situation in 1952.

No proven case of Murray Valley encephalitis occurred in Victoria, and there was no serological evidence that the virus was active in northern Victoria during the summer of 1951-1952. This statement is based on results of serological examination, firstly of persons suspected of suffering from encephalitis, and secondly of normal humans and normal birds in the regions concerned.

From October 1, 1951, to April 30, 1952, sera were sent to Melbourne from 83 patients suspected of having Murray Valley encephalitis. In many instances sera were obtained at least seven days after onset of clinical symptoms. Fifty-five of these suspected cases occurred in Victoria, the remainder in New South Wales and Queensland. None of the sera submitted carried Murray Valley encephalitis complement-fixing antibody; that is, there was no serological evidence that any of the cases was due to Murray Valley encephalitis virus.

During the spring of 1951 it had been determined that certain persons in the Mildura region did not carry Murray Valley encephalitis complement-fixing antibody. At the end of the summer, in April, 1952, sera were again taken from 85 of these previously "negative" persons. A complement-fixation test on the 1952 sera showed that none of the persons had developed antibody during the summer.

During the first four months of 1952, 369 human adult sera were obtained from the towns of Kyabram, Nagambie, Corryong, Tallangatta, Horsham and Dimboola in northern Victoria. Only one serum, taken in Corryong in north-eastern Victoria on April 7, 1952, carried complement-fixing antibody to Murray Valley encephalitis in a titre greater than 1:30. This one positive finding was confirmed by an intraperitoneal baby mouse neutralization test.

We received 287 human sera from New South Wales, collected during the first four months of 1952. Only three showed Murray Valley encephalitis antibody at a titre greater than 1:30. Again the presence of antibody was confirmed by a neutralization test.

Human sera have been received from Cairns, and the area around Brisbane, in Queensland. Altogether 361 have been satisfactorily examined, but only two contained a titre of complement-fixing antibody greater than 1:30. These two came from Atherton and Cairns.

There were thus six sera with a considerable antibody titre taken in 1952—one from northern Victoria, three from New South Wales, and two from Queensland. It is not possible to decide with certainty whether they represent infections with Murray Valley encephalitis in the summer of 1951-1952. It is quite possible, and in fact probable, that the six persons concerned were infected early in 1951, and that their Murray Valley encephalitis antibody had persisted since that time.

Two types of avian sera were collected in 1952. From the earlier work it was known that about 66% of sera from the Little Pied Cormorant (*Phalacrocorax melanoleucos* (Vieillot)) carried neutralizing antibody after the 1951 epidemic.

Therefore sera were examined from six of these birds, hatched in the spring of 1951 and present in Mildura in

April, 1952. As has been recorded elsewhere, none of the six carried neutralizing antibody. It is concluded that there was not a widespread infection among cormorants near Mildura last summer. Fifty domestic fowl less than ten months old were bled in the Mildura region during April, 1952; they were selected from flocks living near the river, where river-bottom mosquitoes were plentiful. None carried neutralizing antibody to Murray Valley encephalitis. Similarly 16 sera from young domestic fowls in Albury were all negative.

If we consider this evidence as a whole, there can be little doubt that Murray Valley encephalitis virus was not active in northern Victoria during the summer of 1951-1952. It is also very doubtful whether the virus was responsible to any appreciable extent for human infection in New South Wales or Queensland. To our knowledge there is no valid evidence for the occurrence of such infections in 1952.

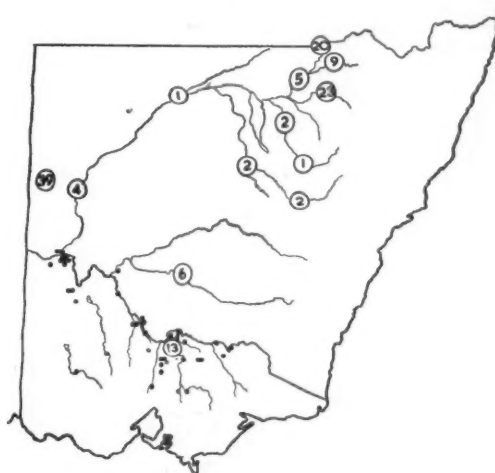


FIGURE 1.
Map of New South Wales and Victoria to show distribution of diagnosed cases of Murray Valley encephalitis, 1951 (black dots), and of X-disease in 1917-1918 (circled figures) from Cleland *et alii* (1918). Reproduced from Burnet (1952).

Before we had obtained this negative evidence, a considerable programme of mosquito collection had been undertaken in the Mildura region. Dr. W. C. Reeves, of California, directed the relevant field work, and his group collected in all over 40,000 mosquitoes during December, 1951, and January and February, 1952. So far Mr. E. L. French has examined over 17,000 of this collection, but has not isolated Murray Valley encephalitis virus from any mosquito. This rather disappointing result is, nevertheless, in agreement with the serological evidence that Murray Valley encephalitis was not active in Mildura during that summer. The work on mosquitoes is to be published in a separate communication.

Meteorological Factors.

The disappearance of Murray Valley encephalitis virus from the Murray Valley in 1951-1952 is in harmony with the history of the disease. In an earlier report we have suggested that Murray Valley encephalitis is probably the same disease as Australian X disease, described by Cleland *et alii*, which occurred in 1917 and again in 1918 in New South Wales and Victoria (Figure 1). In those years it extended to Queensland (Breinl, 1917, 1918; Mathewson *et alii*, 1917; Anderson, 1917). There were two further outbreaks of encephalitis, possibly X disease, on the coast of Queensland in 1922 and 1925 (Anonymous, 1922; Trumpy *et alii*, 1922; Moore, 1922; Baldwin and Heydon, 1925).

Of greater interest for our present consideration is a report of 10 cases of typical X disease in Broken Hill in 1925 (Kneebone and Cleland, 1926). This type of encephalitis is thus known to have been epidemic in western New South Wales and northern Victoria on four occasions over the past thirty-five years—in 1917, 1918, 1925 and 1951—but in the intervening years there has been no sign of the virus.

Miles and Howes (1953) have recently suggested that climatic factors play a major part in the initiation of an epidemic of Murray Valley encephalitis. They have amplified this by an analysis of rainfall figures for northern and eastern Australia, and conclude that excessive rainfall in the preceding spring may precipitate an outbreak. They stress the likelihood that the increased areas of natural water would result in more extensive movement and breeding of water-birds.

water falling in this area drains directly into the Murray River. Further south the catchment area of the Murray River includes rainfall areas numbered 76, 77, 80, 81, 82 and 88, in northern Victoria.

The monthly rainfall for the Darling watershed was estimated as the sum of the figures for the average monthly rainfalls for each of the fourteen districts in the watershed. Monthly figures for this watershed were examined over the period 1913 to 1951 inclusive. There was considerable excess in November rainfall in each of the four years, 1916, 1917, 1924 and 1950, which preceded an outbreak of encephalitis in western New South Wales or northern Victoria. Two of these years, 1916 and 1950, were also distinguished by a heavy excess of rainfall in June and July, but this was not the case in 1917 or 1924.

During the period 1913 to 1951 inclusive, only five years included a November rainfall more than twice the twenty-

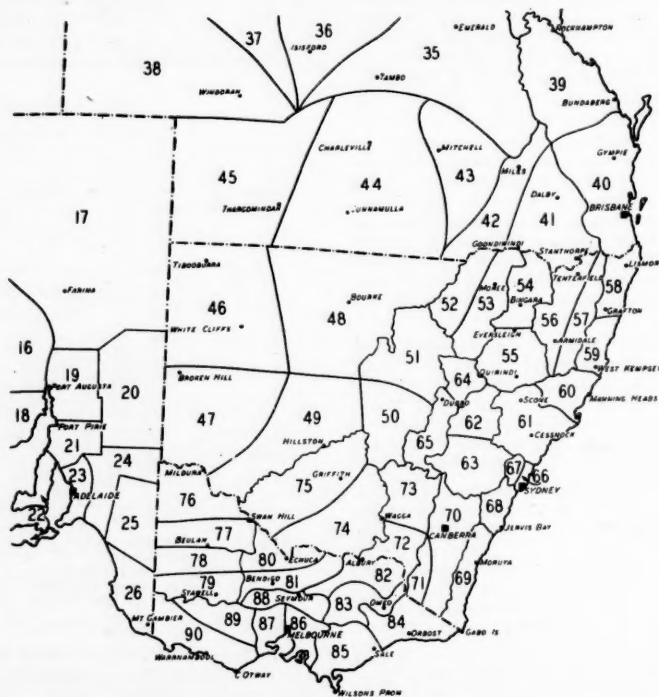


FIGURE II.

Key map to rainfall divisions in south-eastern Australia. Map reproduced by courtesy of the Commonwealth Meteorological Bureau.

Minter (1950) has described the increased winter activity of mosquitoes in central New South Wales following the flooding rains in the autumn of 1950.

From an independent study of the rainfall of the same area, we arrived at a conclusion substantially similar to that of Miles and Howes. Records of rainfall were obtained through the courtesy of the Commonwealth Meteorologist at the Central Weather Bureau, Melbourne. They are recorded as monthly averages of rainfall over districts having a common seasonal regime. These districts, numbered from 1 to 90, are contiguous and cover the Australian continent (Figure II). Areas numbered 41, 42, 43, 44, 46, 48, 51, 52, 53, 54, 55, 62, 63 and 64, in southern Queensland and northern New South Wales, comprise the watershed of the Darling River, the waters from which flow in a south-westerly direction and eventually empty into the Murray River some twenty miles west of Mildura.

Areas numbered 49, 50, 65, 70, 71, 72, 73, 74 and 75, in central New South Wales, are considered to comprise the watershed of the Murrumbidgee, although some of the

four-years mean figure. Four of these were followed by the only four recorded outbreaks of encephalitis in the Murray-Darling Valley. In the fifth year, 1933, the November rainfall was 219% of normal, but we can find no evidence that Murray Valley encephalitis was active in 1934 (Table I).

Rainfall figures for the Murrumbidgee watershed were similarly examined. Again the four crucial years preceding epidemics exhibited grossly excessive November rainfall. The figure for November, 1933, was lower in this region than in the Darling watershed, but in 1934 the November figure was 221% of normal.

In the catchment area of the Murray River in northern Victoria, the situation was different. Certainly there was a high November rainfall in 1916, 1917 and 1924; but in 1950 the November figure was only 119% of normal. Yet in 1951 the epidemic of Murray Valley encephalitis was the heaviest recorded in the Murray Valley. We assume therefore that a gross excess in November rainfall in this

area is not a prerequisite for the spread of Murray Valley encephalitis in this region.

On the other hand, in the Murray catchment area the November rainfall was 268% of normal in 1934, 229% in 1939 and 244% in 1949; yet these three years were not followed by recognized epidemics of encephalitis.

It appears then that the southerly extension of Murray Valley encephalitis virus and its development along the waterways of the Murray Valley in northern Victoria depend not on the local rainfall but rather on the occurrence of excessive rainfall at least 400 miles further north.

TABLE I.
November Rainfall^a as Percentage of Normal.

Year. ^a	Darling Watershed. ^b %	Murrumbidgee Watershed. ^b %	Murray Watershed. ^b %
1916 ..	203	222	223
1917 ..	247	248	205
1924 ..	280	301	315
1933 ..	219	168	155
1950 ..	309	200	119
1951 ..	43	66	57

^a Estimated as described in text.

^b Years preceding epidemics of encephalitis recorded in italics.

^c See text for description.

We believe a reasonable hypothesis is that Murray Valley encephalitis is endemic in Queensland or perhaps in northern New South Wales; and that excessive rainfall in the general Murray-Darling area during winter and again in the late spring may allow the southerly spread of Murray Valley encephalitis from these endemic foci, down into and through the whole of the Murray River basin. Rainfall figures suggest that the crucial area for the excess rainfall may well be the Darling watershed and that one crucial month for excessive rainfall may be November.

However, the absence of encephalitis following just such an occurrence in 1933 is a reminder that an epidemic does not always follow such a meteorological circumstance.

Summary.

The virus of Murray Valley encephalitis spread widely in the Murray Valley early in 1951.

The virus was not detected in the same region in the summer of 1951-1952.

Consideration is given to meteorological factors possibly influencing the appearance of Murray Valley encephalitis in western New South Wales and northern Victoria.

Acknowledgements.

The authors are grateful to all those who contributed to the work reported here, especially to the medical officers and others who forwarded sera during the summer of 1951-1952. We would particularly mention the following: Dr. M. Benson, of Mooroopna Base Hospital; Dr. L. Bryce, Dr. R. Walsh and Dr. T. Golab, of the Australian Red Cross Blood Transfusion Service; Dr. E. H. Derrick, of the Queensland Institute of Medical Research; Mr. N. J. Favaloro, of Mildura; Mr. J. C. Foley and Mr. W. J. Gibbs, of the Meteorological Bureau, Melbourne; Dr. W. R. Horsfall, of the Public Health Laboratory, Cairns; Dr. L. H. Lanyon, of the Mildura Base Hospital; Dr. S. G. Mallarky, of the Microbiological Laboratory, Sydney; Dr. I. J. Mackerras and Dr. M. J. Mackerras, of the Queensland Institute of Medical Research; Dr. D. McLean and Dr. B. Marmion, of the Hall Institute; Mr. B. W. Newman, of the Meteorological Bureau, Melbourne; Dr. J. Tonge, of the Public Health Laboratory, Brisbane.

The authors express their gratitude to Sir Macfarlane Burnet for his invaluable advice during the currency of this study and to Dr. E. H. Derrick for his assistance in the investigation of the early history of Australian X disease.

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Reviews.

The Change of Life and its Problems. By Lillias Blackett Jeffries, M.D., B.S. (London): 1952. London: Victor Gollancz, Limited. 7½" x 5", pp. 136. Price: 4s. 6d.

THIS is an interesting, sensible book. Dr. Lillias Jeffries has a sense of humour, and thoroughly understands the emotions, problems and aspirations of middle-aged and elderly women. She believes that knowledge dispels fear. She explains exactly the physical changes that take place at the menopause in language that can be understood by the average woman. She deals with the problems of the married and unmarried, the busy and the idle, the business woman and the housewife. She has an adequate remedy for nearly every difficulty that could arise. She devotes a chapter to the husband's share, and discusses sympathetically the sexual life of the aging woman. She considers that medicinal treatment has a very small place in the life of the ordinary woman at the menopause. She emphasizes the importance of the right attitude of mind and congenial occupation.

This book is not a scientific treatise, nor a religious tract, but a straight talk by an elderly practitioner, herself a grandmother. If doctors can persuade their patients to read this book, it will save them many weary hours of discussion and admonition.

Side Effects of Drugs. By L. Meyler; 1952. Amsterdam: Elsevier Publishing Company. Distributors: Cleaver-Hume Press. 9" x 6", pp. 280. Price: 30s.

"SIDE EFFECTS OF DRUGS" is the title of a small book by L. Meyler. After reading this summary of the possible toxic effects of modern remedies, a practitioner might well hesitate to use any of them. Actually that is the reason

why this book has been published. The author, a consulting physician at Groningen, in the Netherlands, states that the indiscriminate use of a great number of new remedies has caused so many fatalities and produced toxic effects in such a number of patients that a serious attempt must be made to bring doctors to their senses. This book should be brought to the notice of all medical practitioners. Firstly, the author deals with stimulants such as aminophylline, theobromine, "Cardiazol", "Coramine" and "Benzedrine". He records convulsions, collapse, pulmonary oedema and psychoses among numerous other effects of these drugs. Then he deals with barbiturate acid derivatives including many proprietary preparations. The ill effects of these drugs should be well known; nevertheless the drugs are prescribed as freely as if they were as mild as sugar and water. Papaverine and pethidine, popular analgesics of the moment, are shown to have unrealized toxicity in some cases. The newer anaesthetics, both general and local, are reviewed in the same light. Curare, "Flaxedil" and mephenesin are critically surveyed. Drugs for treatment of Parkinson's syndrome, "Diparcol", "Parpanit", "Artane" and "Myanesin" are critically examined. Fifteen antihistamine drugs which have flowered in profusion almost overnight are discussed in turn and are shown to be almost as dangerous as the conditions for which they are prescribed. The author considers that the antibiotics are most misused at the present time; that there is a tendency to order penicillin, aureomycin, "Chloromycetin" and the rest without due consideration for the need of such potent and potentially dangerous preparations. Histamine injections and hormones come in for criticism also. It would be a good thing if the material in this book could be brought to the notice of every practitioner forcibly and frequently.

Cancer in General Practice. By Ronald W. Raven, O.B.E. (Mil.), F.R.C.S., and P. E. Thompson Hancock, F.R.C.P.; 1952. London: Butterworth and Company (Publishers), Limited. 9" x 6", pp. 274, with 71 illustrations. Price: 48s. 6d.

This book possesses the advantage of being written by a surgeon and a physician in conjunction and contains pertinent advice as to the use both before and after operation of deep X-ray therapy. One good feature is the division into groups of the patients afflicted with cancer. The authors lay great emphasis on early diagnosis with a view to early treatment, and while acknowledging the difficulty of sufficiently early diagnosis, have set out useful methods of routine examination and investigation after diagnosis and treatment have been completed.

Sarcoma of the breast is dealt with; and in the portion of the work devoted to the disease as affecting the stomach, good advice is tendered on the use of gastroscopy, the gastroscope being used in the consulting room without the necessity of the patient's admission to hospital.

The book contains two hundred and fifty pages and has a good index. It also includes a bibliography as well as containing numerous illustrations. The authors are to be complimented on the clarity of their writing and the arrangement of the headings of the paragraphs.

Muscle Relaxation as an Aid to Psychotherapy. By Gerald Garmany, B.Sc., M.B., Ch.B., M.R.C.P., D.P.M.; 1952. London: The Actinic Press. 9" x 6", pp. 66. Price: 5s. 6d. (cloth edition), 3s. 6d. (paper edition).

"MUSCLE RELAXATION AS AN AID TO PSYCHOTHERAPY" is an unpretentious little book by Gerald Garmany. It recognizes the extreme importance of muscular tensions in the causation of so many of the somatic symptoms of anxiety, and in the perpetuation of such symptoms by the formation of the vicious circle of secondary anxiety.

The book is intended primarily for physiotherapists, and the author has wisely included in the first two chapters only a very brief discussion of the role of psychopathology and emotional disturbance, and in the last two chapters a similarly brief discussion of psychotherapy. He properly stresses the view that the role of the physiotherapist is to remain on a physical and not a psychological plane.

The remaining four chapters describe in simple terms the teaching to the patient of the necessary muscle habits, the ability to distinguish between muscle tension and muscle tone, and the ability to continue muscle relaxation when going about the ordinary affairs of life.

It might be doubted whether it is necessary to employ a physiotherapist especially for such a purpose, and both general practitioner and psychiatrist could with advantage teach to many of their patients the lessons of this little

book. They should certainly prove much more effective and healthy hypnotics than the barbiturates that are now prescribed in such enormous quantity for even mild insomnia.

Handbook of Gynaecological Diagnosis for Practitioners and Students. By Walter Neuweiler, M.D.; 1952. London: William Heinemann (Medical Books), Limited. 10" x 7", pp. 462, with 406 illustrations, a few in colour. Price: 80s.

THAT diagnosis in women's diseases is fraught with difficulty and beset with pitfalls is undeniable, and Professor Neuweiler (University of Berne) offers tangible proof of this with his 464-page "Handbook of Gynaecological Diagnosis", intended for practitioners and students.

The first part of the book deals with practical methods of examination. It is an exhaustive survey, and includes manual examination (stress is laid on the combined vaginal and rectal method in examination of the parametrium), examination with instruments (though use of the Sims speculum in the left lateral position is omitted), and special methods. Of these hysterosalpingography is well done and extensively illustrated. Of interest is the insistence on bacteriological control of the cervical secretion even before a Rubin's test is carried out.

The second and main part comprises well-subdivided chapters on diseases of the external genitals, vagina, uterus, adnexa, abdominal wall, and pelvic cellular tissue, with shorter chapters on endometriosis, ectopic pregnancy, leucorrhoea, sterility and low backache. Very comprehensive accounts are found of ovarian tumours, fibroids, cancer of the cervix, adnexal inflammatory conditions and disease of the vulva. The differential diagnosis, however, is often so complete that one loses sight of the important conditions and is overwhelmed with rarities. Much space is also taken by description and diagnosis of advanced malignant disease. The accounts of menorrhagia, metrorrhagia and dysmenorrhoea are very disappointing, while the information in the chapters on leucorrhoea and sterility is too generalized to be of any help.

Though the chapter on low backache is chiefly orthopaedic, far too much stress is laid on gynaecological conditions in the aetiology, and it might well be omitted altogether.

Despite some unimportant errors, such as "insufficient hyperemia", "atrophical", "spermatic veins" (ovarian), the volume is beautifully illustrated and printed, and contains a wealth of information even for the most experienced.

Books Received.

[The mention of a book in this column does not imply that no review will appear in a subsequent issue.]

"A Doctor's Soliloquy", by Joseph Hayyim Krinsky; 1953. New York: Philosophical Library. 8" x 5½", pp. 130. Price: \$2.75.

This is a "confessio fidei" of a scientist writing both in the spirit of a philosopher and of a professional scientist.

"Collegiate Education for Nursing", by Margaret Bridgman; 1953. New York: Russell Sage Foundation. 9½" x 6½", pp. 206. Price: \$2.50.

A critical report on educational qualification for nursing.

"The Practical Management of Pain in Labour", by W. D. Wylie, M.A., M.B. (Cantab.), M.R.C.P. (London), D.A.; 1953. London: Lloyd-Luke (Medical Books), Limited. 9" x 6", pp. 160, with 42 illustrations. Price: 18s. 6d.

The author is an anaesthetist and has as his aim cooperation between obstetrician and anaesthetist.

"Surgery of the Eye", by Meyer Wiener, M.D., and Harold G. Schele, M.D., D.Sc., F.A.C.S.; Third Revised Edition; 1952. New York: Grune and Stratton, Incorporated. 10½" x 7", pp. 469, with 447 illustrations. Price: \$15.00.

A revision of the second edition which appeared in 1949; a prominent feature is the wealth of illustrations and their legends.

The Medical Journal of Australia

SATURDAY, APRIL 4, 1953.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: surname of author, initials of author, year, full title of article, name of journal without abbreviation, volume, number of first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction are invited to seek the advice of the Editor.

MEDICAL WRITING.

In the first paragraph of an attractive brochure on the preparation and writing of medical papers for publication, Dr. W. R. Bett¹ reflects on the ease with which a medical practitioner in the "stately and gracious times of the Renaissance" sat down to write an article which might or might not have been scientifically valuable:

Every advantage was his: he was not embarrassed by too much knowledge; he had no typewriter, no dictaphone, no stenographer, no research secretary—in a less honest age so often a synonym for a ghost writer.

Times have changed and medical writing has become what one might call an endemic disease. Dr. Bett reports that nearly a million scientific articles of one kind and another are published each year, and adds that in spite of this it is the duty of every member of the medical profession to report on worthwhile observations and to interpret his findings. With this we agree. The way in which these reports should be written is a subject of perennial interest.

Another communication, recently published, is entitled "Literary Aspects of Medical Journalism" and is by Harry L. Shaw, who is not a medical man.² This author also quotes some statistics which end with the statement that in the United States about three quadrillion words are published annually, an average of 700,000 words per minute per adult. He refers to these figures in terms of newspapers, novels and so on, and asks us how it is possible for doctors to keep up with the literature in their own field or in a general field. Bett does not adopt such a defeatist attitude. He discusses the problem of keeping abreast of medical literature, and refers to the ways in which a good medical library can be used. In this matter

it is important that those who use medical libraries should learn to help themselves as far as possible. Bett refers to the applicant who expects a librarian to remember "instinctively or by some divine intuition, which article you had read six months previously—in a large green volume, you think, but you cannot recall its title, or the author, or the year". Members of the editorial staff of a medical journal are often expected to have the same instinctive intuition. Here it may be useful to suggest that a practitioner who is interested in a particular subject should keep a notebook in which he jots down references to his particular subject as he comes across them in his reading. Medical readers should not need to be reminded of the value of such publications as The Quarterly Cumulative Index Medicus, the Current List of Medical Literature, and the various abstract journals which appear from time to time. The "Medical Annual" and the yearbooks of the "Practical Medicine Series" are also invaluable to the average reader who wishes to find a reference to a subject. Before we reproduce some of the conclusions set out by Bett, it may be useful to mention some of the more general observations made by Shaw. One of Shaw's important edicts is that all too frequently persons who write, whether they write for medical journals, whether they read or whether they edit medical journals, forget that writing which "does not communicate" is not writing. "A book is not necessarily a book until it is read." He has a great deal to say on simplicity of language and he quotes a statement by Lewis Mumford that "a book has one leg on immortality's trophy when the words are for children and the meanings are for men". He also quotes Lawrence Sterne's view that "writing, when properly managed, is but another name for conversation". The best form of training for a medical writer, or for that matter any writer, lies in the reading of the masterpieces of English literature. Shaw believes that the communication of ideas can be greatly helped by the use of simplicity, by a conversational quality in writing, by writing with concreteness, and by the use of inference. It is often stated by medical practitioners that they themselves are not good writers; they would receive scant consideration from Shaw. He holds that there is no such thing as good writing, that there is only good re-writing. As a professional writer he states that writing is a tedious, laborious, lonely and time-consuming business. Few, if any, men write easily and many of the masters of English literature are known to have written and re-written their works many times before they have been content. Shaw refers also to the man who states: "I write only to please myself." He insists rightly that the trouble with writing to please oneself is that oneself is often only too easily pleased. He has a good deal to say about the titles of articles. He draws attention to the importance of the titles of books and of films in the motion picture industry, and he asks whether "in terms of both typography and wordage" it would not be possible to improve the literary merits of medical journalism by paying a little more attention merely to the title of articles. This is worthy of consideration, not only by authors but also by editors. In scientific literature, a title should indicate the nature of the discussion covered, and should not attempt to summarize the conclusions. Attempts of this kind are sometimes made. When fancy titles are used, they should be used only after careful consideration. Fancy titles cause

¹ "The Preparation and Writing of Medical Papers for Publication" by W. R. Bett, M.R.C.S., L.R.C.P., F.R.S.L. London: Menley and James, Limited. 7 1/2" x 5", pp. 24. Supplied gratis on application to the publishers, P.O. Box 34, North Sydney, New South Wales.

² Arch. Otolaryng., September, 1952.

trouble to the person drawing up the index of a journal, and to a reader who would find a reference to the subject at some time in the future. Some indexers get over this difficulty by indexing the subject and placing the fancy title in parenthesis after it. If medical writing is to be attractive, attention must be paid to its literary quality. Bett holds that the essence of style is the avoidance of wind and of obscurity; and a quality which should be sought is brevity. Bett would regard the term "acute abdomen" as having a definite meaning and as having unavoidably come to stay. In our opinion it is an expression which should be shunned, for it denotes slovenliness of thought. Bett is not sure about such terms as "the doubtful abdomen", "the false acute abdomen", "the traumatic abdomen", "the radiological appendix", "the post-operative stomach", and "the chronic pelvic woman". The last mentioned sounds like a delightful pathological curiosity. All these terms are dreadful. If we allow that there is such an entity as an "acute abdomen", why not a "doubtful" or a "traumatic" one? One rule which may be usefully adopted in medical writing is to avoid the use of a word of, say, three syllables if a word of one syllable can be found to replace it.

Earlier in this discussion, the conclusions stated by Bett were referred to; we quote them in full but do not regard the one marked 2 as essential.

1. Never send the editor a carbon copy of your article, lest he should wonder who has received the original—perhaps a rival editor? Always send the original, but carefully keep a carbon yourself.

2. Use quarto, not foolscap.

3. Type on one side of the paper only.

4. Never type anything in single spacing even though you are sure that the editor will put this in small type.

5. Verify your references.

6. Write; rewrite; rewrite; revise.

7. Make your paper as short as you can. Then make it still shorter.

8. Do not publish the same article elsewhere in a slightly different form.

Another rule which we should like to add for good measure is the following. "Do not think that the whole of one side of a sheet of paper has to be covered; leave a margin and a space at the top of the sheet"; and—yes!—"see that the ribbon on your typewriter is not as old as the typewriter itself".

Current Comment.

DIET AND ATHEROSCLEROSIS.

ONE of the most controversial subjects in modern medicine is the possible relation between the intake of lipids—fats and cholesterol—on the one hand and atherosclerosis and associated pathological conditions on the other. The possibility of this relationship seems to have originated from the fact that cholesterol is deposited in the blood vessel walls in these conditions and the observation that when rabbits are fed relatively large doses of cholesterol they develop atherosclerosis. Cholesterol is not contained in the normal diet of rabbits, so presumably they have not developed an adequate mechanism for dealing with excess of the substance in the body. The work of Rittenberg, Bloch and others has shown that the animal body normally forms cholesterol in large quantities from acetate groups which are derived in the normal processes of metabolism from carbohydrates and

particularly fatty acids. Constantly in relatively large amounts cholesterol is being excreted into the intestine, in the bile and through the intestinal wall. Most of this cholesterol is reabsorbed, but not unless esterified and incorporated in phospholipid-fatty acid complexes. For this process dietary fats are necessary. The amount of cholesterol normally ingested in meat, milk and milk products, and eggs is probably of little importance if one is trying to reduce the cholesterol content of the blood and to prevent the deposition of cholesterol in the tissues in excessive amounts.

It is undoubtedly that in most patients blood cholesterol values can be greatly reduced by severe restriction of total lipid ingestion.

F. Urbach, E. A. Hildreth and M. Wackerman,¹ from available data, arrive at the following conclusions:

1. Restriction of dietary cholesterol alone has no significant effect on human serum cholesterol levels.

2. Restriction of dietary cholesterol and of animal fats has only an occasional—and then not very dramatic—effect on serum cholesterol levels, particularly if the vegetable fat intake is increased.

3. Restriction of dietary cholesterol and of all fat (animal and vegetable) seems to have a profound effect on serum cholesterol levels of normal and hypercholesteremic subjects, provided the total daily intake of fat is limited to 25 grammes or less.

4. Experimentally induced atherosclerosis in animals, and perhaps even spontaneous human atherosclerosis, seem to be reversible, at least to a certain degree.

It is evident that too much importance has been given to low cholesterol diets and many of the diets which have been recommended have such poor nutritive quality as to be actually dangerous.

A new approach to the problem of the relation of lipid intake and atherosclerosis is given by L. M. Morrison.² He has collected data on the incidence of atherosclerosis, or rather of the diseases associated with it, in countries where for a long period, as a result of conditions arising from the last war, the populations have had a low fat content in the diet. In Norway in the years 1940 to 1945 there was a sharp drop in the mortality from circulatory diseases and an almost perfect correlation between the mortality rates for these diseases and the fat intake per head of the population between 1938 and 1948. Deaths from arteriosclerosis, including disease of coronary arteries, in Sweden, Finland and Norway show a large drop between 1940 and 1945, but in the United States where there was little or no reduction in food there has been a steady rise in the number of deaths from these diseases. In England figures from the Medical Research Council reveal that a 50% reduction of mortality from diabetes was observed among patients over the age of forty-five years associated with the marked curtailment of dietary fat during the war years. This reduction was not shown in diabetics in persons under forty-five years of age. It is probable that the reduction in mortality was due to decreased incidence of complicating vascular disease such as atherosclerosis. No figures are available for the present position in England with continuing fat rationing. In France the average mortality rate from coronary thrombosis from 1941 to 1945, inclusive, was 20.6 per 100,000 of population. From 1945 to 1949, after the resumption of normal fat consumption, the mortality rate was 25.5 per 100,000 population. Similar results were obtained in Italy. In all these instances the possibility of coincidence cannot be excluded, but seems very unlikely.

There is little evidence that atherosclerosis and associated conditions are caused by increased intake of fats and cholesterol. Other factors, for the most part unknown at present, are concerned here, and it is probable that there is faulty fat metabolism. The claims by Gofman and his associates that there is an increase in certain fractions of serum lipoproteins in the blood of patients with atherosclerosis are strenuously contested by other

¹ *J. Clin. Nutrition*, September-October, 1952.

² *Ann. Int. Med.*, December, 1952.

workers. Not all patients with coronary atherosclerosis have their well-being improved by restriction in the lipid intake, but an increasing body of evidence suggests that a large proportion of patients show clinical improvement when the fat intake is reduced to a low figure.

FAILURE OF DIURESIS IN CARDIAC FAILURE.

It would be interesting to trace the development of understanding in certain of the more vital aspects of the handling of patients with congestive failure. One of the most significant of such inquiries would traverse the doctrines held concerning that ominous sign, the failure of the renal function, on which we rely for the restoration of the fluid balance and circulation in the anasarctous body. Understanding has been enhanced by the study of that most valuable series of drugs, the mercurial diuretics. Paracelsus would be surprised if he could return to see the great changes wrought by the wider use of mercury, to which we have to bring today a modern acquaintance with the chemistry of those electrolytes that require so nice a balance. It has been common knowledge for some years that after vigorous diuresis in cases of oedematous congestive failure, a syndrome may appear in which the patient suffers from weakness, nausea and mental dulness or aberration, due to a lack of salt. In this low-salt syndrome the level of sodium in the blood is reduced, and usually the serum chloride, with an accompanying reduction in the carbon dioxide combining power. J. H. Stapleton and W. Proctor Harvey have written an article drawing attention to another variant of the electrolyte balance during the administration of mercurial diuretics.¹ They point out that the administration of salt in solution usually reverses the symptoms of the low-salt syndrome, but that another form of acid-base disturbance may follow mercurial diuresis which will not respond to this treatment. The difference between these two syndromes is chiefly biochemical, but clinical acumen should come to the rescue if the amenities of a chemical laboratory are not to hand. Stapleton and Harvey draw a picture of the patient who has failed to respond to a mercurial drug. His signs of congestive failure intensify, diuresis fails, and the customary manoeuvres, such as change of the brand of drug and of the site of injection, are of no avail. If salt is further restricted no improvement follows. Perhaps aminophyllin may produce a better excretion of urine, but sometimes the patient appears to be refractory to mercury, and his condition rapidly deteriorates. The authors consider that at this stage or earlier the serum chloride content and the carbon dioxide combining power should be estimated, and if they are reduced there may be every hope of an irreversible condition being prevented. They remark that it would appear that the kidney is the site of action of mercurial diuretics, which act by inhibiting the reabsorption of sodium or chloride or both in the tubules. Of course it is well known that ammonium chloride will often be effective in starting the renal function again, but it is the authors' thesis that the relationship to the serum chloride of refractoriness of the condition to mercurials should be understood as the chemical basis to a clinical procedure. The occurrence of alkalosis is known to be unfavourable to diuresis in congestive failure, and the significance of the low carbon dioxide combining power now becomes clear. It is not claimed that too sharply defined syndromes can be recognized simply by the figures of one biochemical test, for it is surely likely that considerable differences in the electrolyte levels are present in the body fluids of patients who have incipient or established congestive failure. As Stapleton and Harvey suggest, this may be the basis of individual differences of susceptibility to a refractory state in treatment. If such a state becomes manifest the course is clear. Mercurial drugs must be discontinued, and ammonium chloride administered, in doses of 6.0 to 8.0 grammes daily by mouth, supplemented with dilute hydrochloric acid if necessary. This should be continued for

about a week, so as to build up reserves of chlorides, and, if circumstances warrant, intravenous medication may be used. The importance of caution in the use of diets with a low sodium content in relation to mercurial diuretics is familiar, but the chlorine radical needs some consideration also.

MINERAL OIL.

PURIFIED mineral oil was first recommended for the treatment of constipation by Randolph in 1885. Using the crudest of techniques, he deduced that mineral oil was not absorbed from the intestine and was therefore harmless. Its use was pushed very strongly by Arbuthnot Lane and later by Kellogg of Battle Creek for the relief of the so-called intestinal intoxication. Others followed with claims that it was a complete intestinal antiseptic although "harmless as pure water". All these and other claims were made without any valid scientific evidence.

G. L. Becker¹ has collected the evidence which has been brought forward during the past twenty years to show that liquid paraffin is not the harmless substance it was supposed to be and that it should have little if any place in the treatment of constipation. It has been shown by many workers that it is absorbed through the intestinal wall in sufficient quantities to be demonstrable in the lymph, liver and other tissues. Emulsified paraffin oil may be absorbed through the bowel wall to the extent of 60%, although usually very much less is absorbed. As it is an entirely foreign substance which is not metabolized its absorption can do no good. It interferes to a marked extent with the utilization and retention of calcium and phosphorus. This interference is of dual nature—a mechanical barrier on the wall of the intestine and interference with vitamin D. Another physical effect in the intestines is the coating of food particles with a layer impermeable to intestinal enzymes so that complete digestion and absorption of food is prevented. This effect is further aided by the quickening of the passage of nutrient materials along the tract. Continuous use of mineral oil frequently is associated with severe loss of weight. The fat-soluble vitamins A, D, E and K are readily soluble in mineral oil and their absorption is materially reduced. Lowered prothrombin values are very common in persons using mineral oil over long periods probably owing to the prevention of absorption of vitamin K. The bad effects on the absorption of vitamin A and carotene have been known from the earliest days of research into this vitamin.

A clinical syndrome has been described by Morgan which frequently results from the daily ingestion of mineral oil. It is characterized by indigestion, anorexia, flatulence, fatigue, nervousness, dyschezia and anal leakage. The effects of mineral oil on bowel habits have been summed up as follows. It lubricates the recto-sigmoid and makes an abnormal reservoir of the rectum. The faeces, instead of remaining in the storage area of the colon and sigmoid, leak into the rectum, which remains partially full at all times instead of being empty until just prior to defecation, and symptoms of rectal irritation result. Mineral oil and faeces remain, after evacuation, adhering to the rectal mucosa and are removed with difficulty. Further mineral oil stools are compressed in passage through the anus, forcing an oily suspension of bacteria into the mouths of the anal crypts. Ano-rectal infections follow the administration of mineral oil too often to be coincidental. In one series studied the incidence of ano-rectal infection was almost three times as great in those patients receiving oil as in patients for whom no mineral oil was prescribed. The author sums up as follows.

A survey of the literature before and after its introduction does not disclose a valid reason for the use of mineral oil in the treatment of constipation. On the contrary, the literature contains numerous derogatory reports, any one of which is sufficient to question the advisability of the internal use of mineral oil under any circumstances.

¹ Arch. Int. Med., October, 1952.

¹ Am. J. Digest. Dis., November, 1952.

Abstracts from Medical Literature.

PHYSIOLOGY.

Pulmonary Function Studies.

M. GALDSTON, W. B. WOLFE AND J. M. STEELE (*J. Appl. Physiol.*, July, 1952) report that pulmonary function studies were carried out on 19 patients who did not exhibit clinical or radiographic evidence of lung or heart disease. The patients fell into two groups, made up of eight emphysematous and 11 non-emphysematous individuals, based on the presence or absence of an abnormally large residual volume-total lung capacity relationship. All emphysematous patients also exhibited an abnormally large dead space volume-tidal volume relationship, and six of them some disturbance in either the estimated venous admixture or the alveolar oxygen diffusing capacity or in both. In none was the maximum breathing capacity abnormally reduced. The non-emphysematous patients had a normal maximum breathing capacity. In six the dead space volume-tidal volume relationship was increased, and in all but three there was some abnormality in either or both the estimated venous admixture and the alveolar oxygen-diffusing capacity. The studies indicate that non-obstructive emphysema may occur at a relatively young age; nor is it necessarily an accompaniment of old age.

Hypoxia and Temperature Regulation.

A. HEMINGWAY AND G. G. NAHAS (*Am. J. Physiol.*, August, 1952) report that oxygen consumption rate, respiratory quotient, ventilation rate and rectal temperature of four trained and selected dogs were determined in an experiment consisting of three successive stages in which the animals breathed (i) air, (ii) oxygen-nitrogen mixtures of 6% to 16% oxygen for one hour and (iii) air. During the early part of the hypoxia interval there was a sharp rise in minute volume of respiration and respiratory quotient. Oxygen consumption rate at first fell to subnormal values and then rose slowly to approach normal basal values. In the third period, that is, the post-hypoxic period, oxygen consumption rate was above normal. The rectal temperature fell during the hypoxic period, but rose rapidly after air-breathing was resumed. The results indicate that mild degrees of hypoxia lower the physiological resistance of the dog to cold. This is in agreement with other observations on small animals, such as rats and guinea-pigs, and on man; hence, in general it seems to be a characteristic of homothermic animals.

Influence of Age and Diet Upon Reproductive Senescence.

M. B. VISSCHER, J. T. KING AND Y. CHIUNG PUH LEE (*Am. J. Physiol.*, July, 1952) report that strain A female mice have been studied as to capacity for placentation, delivery of live and dead young at full term, and nursing of young to weaning, in relation to age and to calorie restriction. Reproductive failure with age on the diet

employed *ad libitum* is progressive and is complete as to delivery of live litters at the age of ten months. This occurs with a diet which has been shown to be adequate for reproduction in young strain A mothers through four generations without exposure to any other food. Restriction of calories in the form of lard and dextrose to 50% of *ad-libitum* intake, protein, vitamin and mineral intake being kept constant, for 13.2 and 15.5 months, results in a postponement of reproductive failure to a much later age. If after either 13.2 or 15.5 months of calorie restriction strain A females are subsequently fed *ad libitum*, their capacity to deliver young is appreciably better than that of a continuously full-fed six-months-old mouse. Calorie underfeeding can postpone reproductive senescence well into the last third of the normal life span. Reproductive failure on full feeding of the diet employed occurs by the end of the first third of the life span. Fox chow, a foodstuff with more complex natural ingredients, permits somewhat better lactation than does the semi-purified diet employed mainly in these studies, but it does not yield larger litters or alter the fraction viable at parturition in strain A mice. Senescence rate in physiological characteristics can be altered by the diet in rodents. It appears that intensive studies of these phenomena in mice will be rewarding in terms of principles elucidated.

Effects of Experimental Concussion.

G. W. BROWN, M. L. BROWN AND H. M. HINES (*Am. J. Physiol.*, August, 1952) report the effects of experimental concussion on the common carotid blood flow, arterial pressure and cardiac rate in anesthetized and unanesthetized dogs. Concussion was produced by means of a controlled blow to the occipital area of the animal's head. Experimental concussion is described. Blood flow was measured with an electro-magnetic flow meter. The responses to a concussive blow were (i) loss of corneal reflex, (ii) respiratory arrest, (iii) increased arterial blood pressure, (iv) increased blood flow through the common carotid artery and (v) development of cardiac arrhythmia. The cardio-vascular effects of a blow to the head were short-lasting, all significant changes occurring within the first fifteen minutes following a concussive blow. Since the cardio-vascular response pattern of dogs is similar in electroshock, concussion, subconcussive blows and head blows resulting in skull fracture, whether or not the animal is under general anesthesia, it appears that this response pattern is not unique, but may be evoked by various stimuli of a more or less traumatic nature. This study offers indirect evidence in support of the contention that the "loss of consciousness" frequently associated with clinical concussion is not due to a diminution in cerebral blood flow.

Evaporation from the Skin.

A. B. HERTZMAN, W. C. RANDALL, C. N. PEISS AND R. SECKENDORF (*J. Appl. Physiol.*, October, 1952) report that the rates of evaporation of water from the skin of the face, hand, foot, arm, leg and trunk were separately measured by the desiccating capsule technique, in resting nude subjects who were exposed to environmental temperatures in the

range of 24° to 38° C. At temperatures below the sweating thresholds, insensible perspiration proceeded at a fairly uniform rate (six to ten grammes per square metre per hour) from the skin of the arm, leg and trunk. Palmar, plantar and facial rates were higher (up to 100 grammes per square metre per hour) for the palm. The onset of sweating occurred in the various skin regions at different environmental temperatures, thus implying a recruitment of sweating which ascended from the distal portion of the extremities to the higher levels of the body as the evaporative demands increased. The possible mechanisms of this phenomenon are discussed. The regional fractions of the total cutaneous evaporation demonstrated domination of evaporative regulation by the legs at temperatures slightly above the sweating thresholds. At temperatures above 34° C., increases in sweating were linear with the rise in temperature and about equal over the body surface. The continued domination of evaporative heat losses by the leg and trunk resulted from the mass effect of their extensive surfaces.

Effects of Hypoxia on Pulmonary Circulation.

B. M. LEWIS AND R. GORLIN (*Am. J. Physiol.*, September, 1952) report that the cardiac output and pulmonary and femoral arterial and left atrial pressures were measured in 16 dogs during the inhalation of gas mixtures containing 2.5% to 10% of oxygen. Twenty-four experiments were performed in nine dogs with 10% strength oxygen. The results may be divided into two groups. (a) Nineteen experiments in which arterial oxygen saturations were above 55% showed an increase in pressure gradient across the lungs but little change in cardiac output and, over-all, a statistically significant rise in pulmonary vascular resistance; this response, however, was not invariably observed. (b) Five determinations in which arterial oxygen saturation was below 55% showed uniformly a rise in cardiac output and a fall in pulmonary vascular resistance; this is similar to the cardio-vascular response in acute severe hypoxia. Twelve experiments were performed on seven dogs with 2.5% to 4.7% strength oxygen. Arterial oxygen saturation was 27% or lower. Pulmonary artery pressure increased in 11 experiments. When arterial oxygen saturation was 15% or less, left atrial pressure increased. Cardiac output rose if severe hypoxia was very brief, but fell if it was prolonged. Pulmonary vascular resistance fell in 10 of 12 experiments. The Fick principle is felt to be applicable to the determination of mean cardiac output during changing states and at low arterial oxygen saturations when integrated simultaneous measurement is made of arterio-venous difference and oxygen consumption. Mild or localized hypoxia causes no change in the cardiac output and is associated with pulmonary vasoconstriction. This reaction is probably due to the local action of low oxygen tension in the pulmonary vessels. Severe hypoxia causes an increase in cardiac output and is associated with a decrease in pulmonary vascular resistance. This observation may be related to (a) lowered venous blood oxygen tension, (b) accumulation of metabolites in the blood, or possibly

(c) reflexes acting through the vasomotor centre, or (d) sympathico-adrenal stimulation.

BIOCHEMISTRY.

Fibrinogen.

L. LORAND AND W. R. MIDDLEBROOK (*Biochem. J.*, October, 1952) have treated bovine fibrinogen and fibrin by the dinitrophenyl method and determined the N-terminal residues. In the fibrinogen molecule two chains end in tyrosine and one in glutamic acid. The same unit weight of fibrin has two N-terminal residues of tyrosine and four of glycine. It was shown that the action of thrombin on fibrinogen results in the fission of glycol-peptide bonds within the molecule, with the formation of fibrin and, it is suggested, the simultaneous removal of N-terminal residues of glutamic acid as part of a peptide. Fibrinogen and fibrin contain the same number of free ϵ -N groups of lysine.

L. LORAND (*ibidem*) has also shown that the clotting activity of thrombin, known to be a specific protease, is accompanied by the liberation of non-protein nitrogen. A peptide appears in the fibrinogen-thrombin system. It is suggested that this hitherto unknown substance should be called "fibrinopeptide", since it is believed to be derived from the splitting off of part of the fibrinogen molecule.

Milk Proteins.

P. N. CAMPBELL AND T. S. WORK (*Biochem. J.*, October, 1952) have used valine, lysine and glycine, each labelled with ^{14}C , to investigate the synthesis *in vivo* of milk proteins in the rabbit. They have shown from the activity/time curves for the milk and plasma proteins that under the conditions of these experiments the blood amino acids contribute most of the nitrogen for the synthesis of the milk proteins. A comparison of the specific activities of the labelled amino acids isolated from the radioactive milk proteins indicates that there exist at least two forms of precursors contributing amino acid residues for the formation of milk protein, namely, amino acids and peptides.

Cortisone.

M. J. H. SMITH (*Biochem. J.*, December, 1952) has demonstrated that salicylate reduces the glycosuria and hyperglycaemia induced by cortisone in the normal rat. Cortisone causes deposition of liver glycogen in the adrenalectomized rat, while the concurrent administration of salicylate not only produces depletion of existing glycogen but also prevents the deposition of new glycogen by the cortisone.

Bone Metabolism.

T. F. DIXON AND H. R. PERKINS (*Biochem. J.*, October, 1952) have reported that the citrogenase and aconitase activities of various rabbit bone regions are relatively greater than that of *iso*-citric dehydrogenase compared with kidney and liver. Increasing concentrations of citrate inhibit the calcification of hypertrophic rat

cartilage *in vitro*. Enzyme levels in bone indicate the relatively greater metabolic activities of metaphysis and epiphysis over that of cortex. Amounts of citrate in bone regions are in inverse ratio to their metabolic activities. It is suggested that the higher "citrogenase" and lower *iso*-citric dehydrogenase levels in bone regions may produce local increased concentrations of citric acid, which may then become coprecipitated during deposition of bone salt.

Blood Urea.

H. L. KORNBERG AND R. E. DAVIES (*Biochem. J.*, October, 1952) have reported that when ^{15}N urea was injected subcutaneously into a 2.0-kilogram cat, 2.5% of the ^{15}N was excreted in the urine in forms other than urea. The tissues contained nitrogen with slight but significant excess of isotope. The fate of ^{15}N ammonia in the body was studied by measuring the distribution of ^{15}N in the tissues and urine after subcutaneous injection into a 1.2-kilogram cat of ^{15}N ammonium lactate in ten four-hourly doses. Of the nitrogen of the injected ammonium lactate 54.2% was converted to ^{15}N urea. It follows that in forty hours at least 5% of the urea of the cat was broken down in the body. After administration of ^{15}N urea or ^{15}N ammonium lactate the nitrogen of the gastric mucosa was not preferentially enriched with ^{15}N ; this argues against the participation of gastric urease in local synthetic mechanisms.

Folic Acid.

E. C. NABOR *et alii* (*Arch. Biochem.*, May, 1952) have examined the effect of folic acid and vitamin B_{12} on glycine toxicity. Folic acid was found effective in reversing the toxic effects of glycine. Folic acid determinations on the livers of chicks receiving glycine indicate an increased storage of the vitamin. The total creatinine content of blood, muscle and liver is higher in folic acid-deficient birds.

Urinary Ammonia.

B. M. A. DAVIES AND J. YUDKIN (*Biochem. J.*, November, 1952) produced changes in ammonia excretion in rats by the induction of chronic acidosis and chronic alkalosis. After three to eight months, some selected renal enzymes were assayed in these animals by determining ammonia production by kidney slices, from *L*-glutamine, glycine, *L*-leucine, *L*-aspartic acid and *L*-alanine. These substrates were selected to investigate possible adaptation of four enzymes which might be concerned in ammonia production: *L*-glutaminase, glycine oxidase, *L*-amino acid oxidase and the transaminase system. Chronic acidosis resulted in an appreciable increase, and chronic alkalosis in an appreciable decrease in ammonia production from *L*-glutamine, glycine and *L*-leucine. There was no significant change in ammonia production from *L*-aspartic acid and *L*-alanine. These results suggest that urinary ammonia is produced, at least in part, by (i) deamination of glutamine, (ii) deamination of glycine by glycine oxidase, and (iii) deamination of other mono amino monocarboxylic acids by *L*-amino acid oxidase. Reasons are given for supposing that deamination of *L*-alanine is effected chiefly, if not entirely, by the transaminase system which also

acts upon *L*-aspartic acid. It would appear either that this system plays no part in the normal mechanism of urinary ammonia formation, or that one or more of its component enzymes is not adaptive. The adaptive changes found in three renal enzymes indicate that acidosis and alkalosis cause a change in ammonia formation and not merely in ammonia elimination.

Prostate Gland.

C. A. MAWSON AND M. I. FISCHER (*Arch. Biochem.*, April, 1952) report that the posterior prostate of the rat contains about the same amount of carbonic anhydrase as erythrocytes, whereas the ventral prostate of the same animal contains a negligible amount of this enzyme. The lateral portions of the posterior prostate contain six times as much zinc as the median part, and they also contain two to six times as much carbonic anhydrase. The presence of zinc in the prostate cannot entirely be accounted for by the zinc-containing enzyme carbonic anhydrase, which is practically absent from rabbit prostate. The proportion of zinc to enzyme in rat posterior prostate is greater by a factor of five to ten than it is in the red cell, where all the zinc is associated with carbonic anhydrase.

Vitamin B_{12} .

J. W. DUBNOFF (*Arch. Biochem.*, May, 1952) has presented evidence that an *Escherichia coli* mutant, which requires vitamin B_{12} or methionine, can grow and therefore synthesize methionine in the absence of vitamin B_{12} if homocysteine or certain reducing agents which can reduce homocysteine are present. The growth-promoting value of homocysteine is enhanced by vitamin B_{12} . The trace requirement of vitamin B_{12} may be replaced by the methyl donor, dimethyl- β -propiophetone. The latter is active only in the presence of the reduced methyl acceptor. It can also be replaced by catalytic amounts of *p*-aminobenzoic acid. This suggests that *p*-aminobenzoic acid synthesis is impaired in the absence of vitamin B_{12} and that vitamin B_{12} does not take direct part in the synthesis of the methyl group *per se*. The data implicate vitamin B_{12} in maintaining homocysteine in the reduced state. They exclude vitamin B_{12} as a coenzyme for transmethylation in this organism.

Vitamin D.

S. A. BELLIN AND H. STEENBOCK (*J. Biol. Chem.*, January, 1952) have shown that the urinary citrate excretion of both young and adult rats was increased by the administration of physiological as well as excessive amounts of vitamin D. This effect was obtained with young rats which had been made rachitic on low phosphorus rations, with rats on low phosphorus rations made alkaline with sodium bicarbonate or potentially acidic with ammonium chloride, and with rats kept on a non-rachitogenic ration containing adequate amounts of calcium and phosphorus. While the level of citrate elimination was highest in rats on the low phosphorus rations, the increases due to vitamin D were highest in those on rations which contained phosphates. With low phosphorus rations, dietary calcium also increased the urinary citrate content,

Special Articles for the Clinician.

(CONTRIBUTED BY REQUEST.)

LIX.

ANXIETY STATES.

"Canst thou not minister to a mind diseased,
Pluck from the memory a rooted sorrow,
Raze out the written troubles of the brain. . ."

MACBETH, Act V, Scene III.

THE anxieties arise as an expression of conflict and are a basic physiological or protective mechanism indicating distressing or unpalatable circumstances. Anxiety presents in all degrees of intensity, from a state of mild excitement and over-awareness to one of acute galvanizing terror. Likewise each system can be affected in various degrees of intensity—the response of each of these systems varies to some extent with the individual's susceptibility and with the duration of the condition. The complaint as described by the patient may appear to be confined to one organ or one system, but inquiry will very soon reveal the wider distribution of symptoms that is typically seen in a functional illness.

In this respect the expression "target symptoms" has been used to suggest the central origin of the condition with a disturbance in distant organs—on the analogy of an artillery piece dropping shells now on this point, now on that. Up to a point the combination of symptoms is individually variable, although the underlying pattern is clear for diagnosis.

It seems probable that the reactions which produce these symptoms in the tissues are in the beginning reversible, but if they remain present for a long period some organic change is often associated, and the target symptoms become an organ disease, as is quite evident in the emotional factors and tensions that antecede peptic ulcer and asthma, for example.

It is instructive to observe how our bodies respond in a uniform way to physical and psychological stimuli, even though these may occur years apart. In this respect it is very helpful, in determining the presence of a functional disease, to inquire carefully whether there have been similar episodes in days gone by. It will often be revealed that there have been one or more episodes with symptoms similar to those now presented. A direct question, of course, may produce a negative answer, but if symptoms are discussed—their mode of occurrence, association with other symptoms—a well-established pattern of response becomes evident.

There are three main ways in which anxiety may be classified, namely:

1. Environmental stress, due to increased responsibility, or emotional stress associated with work, home, or other obligations.
2. Limitation of ability to handle a situation or emotion, due, for example, to lack of training or limited experience in a situation—increased sense of duty or responsibility as seen in conscientious or scrupulous persons.
3. Constitutional and probably biochemical limitations, for example, after some debilitating illness such as influenza, premenstrually, *post partum*, or spontaneously in some mood depression.

In the first two of these, namely, those conditions involving environmental stress and associated with limitations of ability, there is a conscious awareness of the distress and to some extent of its cause. It would seem as if Nature was trying to impress the individual with the sense of conflict then present, and it may be that the persistence of the symptoms is indicative of the natural demand for a resolution of the emotional discord.

By way of contrast the constitutional or biochemical form has fundamentally no environmental basis, and although the patient may think back and even invent an explanation, it is clear to the outsider that this rationalization cannot bear critical examination, and is in fact no explanation. It becomes further evident that there is no unusual environmental change and the condition arises as a spontaneous expression of illness—an illness whose origin we do not, with our present knowledge, satisfactorily understand. It should be noted that these divisions are quite arbitrary, and there is no reason why more than one cannot exist simultaneously.

To be more specific then, it may be stated that anxiety is treated by any doctor in his practice—it need not be handed over with kid gloves to a specialist or psychoanalyst. First, therefore, if one is to do oneself justice and to avoid unnecessary repetition it is wise to set aside at least an hour for the history taking. As necessary as seeing the patient is an interview with some relative or person who can further elaborate the history. The patient, too, has a feeling of completeness if he has talked himself to a standstill. Some—the obsessional type, who tell the story with photographic detail—must be stopped after a fair hearing in the interests of one's own nervous state. When the spontaneous stream is lagging, one may ask for detailed description of symptoms which will make it quite clear that these are functional disturbances, for example, the functional headache, the tight band, the pain in the head unremittingly present for days or weeks, unrelieved by aspirin, aggravated by emotional stress, relieved by sedation and rest; or the dyspepsia, unrelated to food, unrelieved by alkali, and associated with much flatulence. The symptoms of the straight-out anxiety—headache, anorexia, loss of weight, tremor, difficulty in going to sleep, anxiety dreams (of trying situations), fatigue, irritability—are all well known.

In cases in which the environmental situation is considered the main factor, I am certain it is important to let the patient make up his own mind as to what adjustment he is prepared to make. Has he developed a sufficient feeling of injustice to be really prepared to leave home, or does he simply want a sympathetic listener who will hear all his troubles and will oblige in return with a few words of encouragement and understanding explanation? It is wise in this event to counsel patience and recommend that no important decision be taken when the patient is upset, and to prescribe some sedation, to be taken during the day and in order to enable him to go to sleep if necessary.

The vast majority of these environmentally produced anxieties will resolve themselves in time or will reach such a degree of compromise and resolution as the persons involved are willing to allow at this time. For to them the conflict may be well nigh unsolvable, for example, marital conflict *versus* financial dependence and the responsibility of the care and upbringing of children.

Conversion hysteria, or an anxiety converted into a somatic symptom, can be both simple and exasperating. In one light it is an escape through disease. If the condition is fairly easily diagnosed, for example, hysterical aphonia or *dermatitis artefacta*, it is important not to cure the symptom before the reason for the sickness is thoroughly understood; otherwise one is likely to be faced with a more complex syndrome a few days later. The approach should be to relieve the need for symptoms, as is done with the straight anxiety, either by altering the environment or by helping the patient to adjust to the circumstances.

The obsessional and compulsion neuroses occur together and can be dealt with together. This condition presents most frequently in the chronic worrier or fussy, constantly aware of a feeling of incompleteness. Tension is created if some absolute standard of achievement is not reached. The conscientious person becomes over-conscientious and hyper-critical of himself; by this time a condition of emotional depression can be recognized. There may be worries about responsibilities at work and at home, about religious scruples, germs, cleanliness and all manner of phobias. The patient can see that these beliefs are unfounded, but nevertheless is compelled to perform some ritual for the relief of tension.

It is clear that these obsessional and compulsive states are but an exaggeration of the patient's previous personality. Attention can best be directed towards relieving the emotional vector, which is usually some form of endogenous depression and will be dealt with now.

Depression is of two main types—reactive and endogenous. By reactive is meant the state that follows a disappointment or tragedy in one's life. In a comparatively short time, days or weeks, an adjustment of sorts is made, and this improves as time goes on. It calls for a little sedation and understanding, some occupation and a removal if possible from disturbing surroundings.

Endogenous depression (cyclic depression) is probably biochemical in origin. It is relatively common and rightly a dreaded disability. It may arise spontaneously or follow some mild intercurrent infection—or endocrinal disturbance. It is characterized by a lack of energy and interest, a loss of confidence and tendency to look upon the worst side of everything. There are in addition a depression of all bodily functions, loss of appetite, loss of weight, inability to concentrate *et cetera*. Endogenous depression differs from the

other form of depression in that it is more profound and more prolonged, and may reach such a degree that suicide seems to the patient the only logical course. This condition may last for months, varying in intensity, usually worse in the morning, sometimes slightly improved towards evening. After an unpredictable period of time, a spontaneous remission can be expected. Sedation for both day and night is often helpful—the depressed patient will often say that the only peace he gets is when he is asleep. It is wise therefore not to prescribe too large a quantity of sedatives. Amphetamine is sometimes useful taken in the morning and at lunch time. If the depression is profound or prolonged electric shock treatment will give a respite. It does not cure the illness, but it relieves the intensity of the depression; it may have to be repeated if the intensity of the depression should increase subsequently. Unlike the reactive depression, there is no relief to be obtained by change of environment; in this instance the patient takes his misery with him wherever he goes.

In reference to sedation there are a few points worth mentioning. First, it is always necessary to give sufficient sedation to produce the desired effect—whereas phenobarbital half a grain twice a day may sometimes be necessary to give relief of symptoms, at other times one grain three times a day will be needed. "Amytal" one and a half grains twice a day, when necessary, may be useful in states of panic or acute tension; sometimes it may be desirable to give "Sodium Amytal" three grains three times a day. Bromide is useful during the day, up to 15 grains three times a day; so also is chloral hydrate in one of the standard mixtures, from seven and a half grains to fifteen grains twice or three times a day.

The second important point is to realize that individual patients have different susceptibilities to drugs—it is wise to ask the patient what has been prescribed formerly and what gives him relief. It is just as important to elicit what not to prescribe. Most physicians have their favourite night sedative, and I do not think that one group of drugs is very much better than another. Depth of sleep and the early or late action of the drug are also points worth consideration.

Before concluding mention should be made of psychoses that occasionally present as an anxiety state. Particularly interesting is the young person who quite suddenly awakes at night in a state of panic and without any reason may perhaps be fearful of dying that night—the whole condition resolving after a day or so, usually with the help of some sedation. As a rule no obvious cause or satisfactory explanation is to be found for such an episode. It has been my experience that this is frequently the early manifestation of a schizophrenic psychosis. There may be several more of these episodes, months or years apart, before their psychotic nature becomes evidence.

One cannot overlook either the psychotic who has delusions, but with some insight—perhaps ideas of persecution; a system is being worked; there are spies and hidden microphones and his thoughts are being intercepted—the result being that the genesis of the anxiety is not revealed. If one is aware that such states do occur, one is likely to pay heed to any casual remarks which may supply the leads for more direct questions.

We should remember that a triangular situation may exist between the doctor, the patient and his relatives, and the illness. All too frequently if the patient does not get better, the doctor is likely to blame the patient for his illness, or to imply that he is not trying, or does not want to get better. This attitude of self-righteousness is altogether bewildering to a patient suffering from a functional disturbance. One should remember also to be very careful not to be misunderstood by the patient. The doctor may say that there is nothing organically wrong with the patient, meaning to convey that there is no detectable dysfunction in any particular organ of the body. Yet every doctor knows that there is a very real difference between feeling well and being well—the first implies the part and the second implies the reality as well as the feeling and emotional adjustment to this state.

Perhaps the surest way that we can assist our patients is to be helpful with their troubles. The outpourings that seem so tedious and are so often repeated are just the means of letting down the emotional tension. It is, I think, by this supportive therapy (the sympathetic ear, the encouragement) that we as a profession have acquired a position of privilege, and not, I fear, by the science that we spent so long acquiring and pursue with so much diligence.

G. B. MURPHY,
Brisbane.

Medical Societies.

SYDNEY INSTITUTE FOR PSYCHOANALYSIS.

THE annual meeting of the Sydney Institute for Psychoanalysis took the form of a symposium on the psychotherapy of obsessional neurosis. It was held on September 10, 1952, in the William H. Crago Council Chamber, British Medical Association House, 135 Macquarie Street, Sydney.

Dr. R. C. WINN, the chairman of the institute, briefly referred to its activities during the first year of its existence. He said that the main function of a psychoanalytical institute was the training of psychoanalysts, and that two candidates were taking the course. Another educational aim of the institute had been met by providing two courses of seminars, one for medical graduates, the other for psychologists and social workers; also the nucleus of a psychoanalytical library had been formed. A separate department of the institute was a psychoanalytical clinic where 70 consultations had been conducted.

The Obsessional Neuroses.

Dr. CEDRIC SWANTON, discussing the obsessional neuroses, said that the treatment was a very difficult business. As Freud had once said: "The problem of the compulsion neurosis is as yet unsolved and as such it remains unconquered." Dr. Swanton was sure that they were all fully aware of the immense difficulties associated with the treatment of the condition by psychoanalysis. To enumerate, very briefly, only a few of those difficulties, he might begin with the theory that in the obsessional neurosis there was a regression to the anal sadistic level. That gave rise to a grossly ambivalent and bisexual transference situation, and they might find that the patient tended to become completely absorbed with the transference situation. Further, there was a very real tendency on the part of the patient to counter the analyst's interpretations by theorizing on his own illness. Superficially, that passed for cooperation, but it really meant another defence by a change of symptoms. The patient flew into his own little magical world of concepts and words, and he isolated himself from subjective feeling. Again, he had great difficulty, of course, with free association. His over-conscientiousness was such that it became an obsession. Free association became merely a meticulous recounting of experiences, interspersed with all sorts of hair-splitting and over-compensatory cooperation in the wrong direction. Still further, thought and speech, which were the instruments of psychoanalysis, were themselves affected by the obsessional neurosis. One patient of Fenichel's had said that it seemed to be something like falling into the water with a towel in one's hand and then attempting to dry oneself with the wet towel—surely a surprising piece of insight on the part of the patient. Lastly, the analyst might not only represent the patient's super ego, but he might appear as a seducer of the "good" obsessional, of whom the patient became afraid, and naturally the patient fought the analyst as hard as he could. The whole thing tended to become intellectualized, the analysis tended to bring out the analyst's own latent obsessionalism, and the analysis might become an obsession in itself. There were, of course, obsessional traits of greater or lesser degree in all human beings, just as there was obsessionalism in all the ritual of living, and one might even extend that to the periodicity and rhythms of life itself. Dr. Swanton ventured to say that there was something of obsessionalism in psychoanalysis itself, with all its regularity and countless repetition in search of the ultimate perfection and the universal demand for a rebirth. As an instance of that, a colleague recently returned from England had told him of the analysis of an obsessional which to his knowledge was now in its sixteenth year. Another case was in its sixth year. In those cases, one wondered whether it was the analyst or the analysand who was now the patient. No doubt the analyst could rationalize those interminable procedures on the basis of supportive treatment. Dr. Swanton said quite seriously that the analyst was quite likely justified in that rationalization.

Dr. Swanton went on to say that the cases to which he referred were the "true blue" obsessional neuroses, which had usually commenced early and persisted. There were, of course, minor obsessional illnesses occurring in later life with obvious precipitating factors that might clear up more quickly. However, in general, he would say that, as he did not know of any other form of therapy that was not physically destructive (for instance, leucotomy, whether by knife or by electricity), one should give the patient the

chance of undergoing analysis. The uncomplicated obsessional neurosis did not respond to electric convulsion therapy. One of the things about the condition that interested him most was the reason why the illness was so intractable. They knew most of the analytic difficulties, but then they might be wrong in assuming that analysis was the ultimate and universal answer to the cure of the condition. It seemed right in theory, but it did not always work out in practice, and they would do well to remember one of Freud's more famous remarks to the effect that "all this edifice of psychoanalytic theory will one day have to be placed on its physiological foundations".

Dr. Swanton said that he could not help feeling that one of the major factors in the irreversibility of the obsessional disorder was that it was based on the obsessional personality and tendencies, and that they had very strong genetic or diathetic factors in the production of that type of personality. They did see, for instance, the Mussolini type of baby—the short and thick and usually dark, resistive, negativistic, active, restless, obstinate, stool-holding variety of infant. They were not easy babies, and those characteristics could be observed at six months or even earlier. Then analysts saw all the offshoots of the true or pure obsessional neurosis. They saw the very pronounced association of obsessional characteristics in all the manic-depressive types—so much so that if he could not have it confirmed that a depressive was not only normally a bright, cheerful, active and energetic sort of individual, but that he was also tidy, clean, particular, methodical, punctilious, over-conscientious, over-scrupulous, over-meticulous and so on, he began to doubt his diagnosis. Dr. Swanton did not think that there was much doubt about the genetic factor in the manic-depressives; one often saw not only obsessional characteristics, but an associated and accompanying obsessional illness with mood disturbances. One saw the obsessional disturbance clear up dramatically with the depression, under physical methods of treatment. Again, one saw the obvious genetic or diathetic factor in the stammerers, with the almost ludicrous repetition and similarities of personality in all such children and in one or other or both parents, and to a lesser extent in the migraine sufferers and others. He wondered whether that diathesis meant a physiological predisposition with facilitated central pathways for the obsessional means of protection by the intellectualizing of emotional factors, with a sort of cortical divorce of intellect from emotion—actually a sort of compartmenting of the intellectual and the feeling levels. It was a different sort of dissociation from the schizophrenic with no disruption, but full preservation of intellect and cortical thinking and understanding.

Dr. Swanton said that it fascinated him to speculate on the physiology of the obsessional neurosis in terms of inherited facilitated cortical patterns or circuits, and dissociation based on the ideas of engineering transmission such as those used in the theories of cybernetics. One could perhaps picture those rituals and compulsions induced by means of cortical inhibition, resulting in reverberating and closed circuits and so on. Actually, such obsessionals performed a kind of leucotomy on themselves, and rather than cut off the cortex from the thalamus, one had the feeling that one would like to bore holes in that artificial partition and allow a freer mixture to occur. However, the underlying destructive aggressiveness of the patients would no doubt destroy them if released.

Dr. Swanton went on to say that his experience of surgical leucotomy in the obsessional neuroses was too limited to be of value. He had not had a great deal of success, although he had had a couple of surprising, although perhaps temporary, successes with electrical leucotomies. His impression of leucotomy in the treatment of obsessionals was that they were considerably relieved if there was a great deal of tension associated with the condition, but that if there were a lot of expiatory acts, compulsions and rituals, those tended to persist. Such obsessionals had to some extent leucotomized themselves, and it would seem that a leucotomy completed the job of severing the connexion between the intellectual processes and their association with subjective feeling. Incidentally, he had recently reviewed some patients who had undergone the operation of leucotomy some years previously and who appeared to have benefited very considerably at the time. Although symptomatically they were better, he could not help feeling uneasy at the changes in the personalities of the patients. They were not the same people, and one felt that although they were able to "fit in" and live socially, they were lacking in rapport, they seemed vaguely isolated and did not appear to be quite "with us".

In conclusion, Dr. Swanton said that, despite all the immense difficulties associated with the procedure, he felt that psychoanalysis must, at the present time, be the treat-

ment of choice. Unfortunately it was only too frequently impracticable because of the economic factor. Failing it, one could only use the psychotherapeutic understanding one might be blessed with and support the patient and attempt to relieve his tension through the positive transference. The tension in gross irreversible conditions might be relieved by the operation of leucotomy. By and large, unfortunately, he thought that Freud's early remark quoted at the beginning of his paper still held good: "The problem of the compulsion neurosis is as yet unsolved and as such it remains unconquered."

Obsessional Neuroses in Children.

DR. IRENE SEBIRE discussed obsessional neuroses in children. She said that there was fairly general agreement that most children indulged in some form of magical practice, or observance of significant ritual, or believed in the efficacy of charms to ward off bad or encourage good luck. Group games, governed by rigid rules, with the development of symbolic expression, were typical of shared play activities in the early period of latency. There were certain observances to be respected, definite prohibitions to be honoured, as the child learned to control his primitive drives in conformity with group requirements. The use of signs or rituals seemed to establish a kind of insurance against impending threats, or a guarantee of achievement and satisfaction of desires. Those relatively common practices in childhood were not in themselves abnormal, unless a refusal to respect them, or an interruption or interference with particular technique brought recurring and extreme anxiety to the disturbance of the child's peace of mind. Such anxiety might lead to inappropriate or exaggerated patterns of behaviour in the form of later compulsive or obsessional disorders.

Age of Onset.

Melanie Klein had stated that isolated obsessional traits which emerged in the first period of childhood were not organized into what were later regarded as obsessional neuroses until the beginning of the latency period. At that stage the more mature ego elaborated and synthesized those obsessional characteristics which had been present since early childhood, although not clearly recognizable.

Some writers suggested that obsessional neuroses were not diagnosable as such until the end of the latency period; others suggested that they did not appear in definite form until puberty was established. Professor Kanner quoted the opinion of several observers that approximately 50% of adult cases could be traced back to childhood; others held that the onset was insidious, its symptoms apparently dating from a precipitating crisis or shock, when they merely become more severe. Professor Hadfield was of the opinion that from two and a half to three years was the usual time for establishing the basis of obsessional neurosis, particularly when the child was replaced by another sibling at that age. Bender and Schilder used the term "impulsions" to describe a disorder in the behaviour of children whose interests, ideas and actions, and preoccupation with phantasies regarding a specific object, were engaged in with considerable eagerness and satisfaction. Difficulties arose when persistence of that interest interfered with their ordinary activities and environmental relationships. They believed that "impulsions" were more characteristic of childhood, while obsessions and compulsions belonged to adolescence and adult life. At about the age of ten years, the ego and superego took a definite stand regarding such impulsions, when they became either compulsions or so-called obsessional neurotic character traits. In the opinion of Bender and Schilder, Melanie Klein's references to obsessional neuroses in children might have referred to cases of impulsions, which they considered could be the result of transformations rather than the direct expression of various drives. According to their concept, the impulsion could be the nucleus for the development of a later obsession. There seemed to be no general agreement as to the part hereditary predispositions played in the creation of obsessive or compulsive characteristics. On occasion one found that, although parents had not been concerned at earlier compulsive tendencies, recognition came when a precipitating factor as a shock, a disappointment or too demanding a situation set in train activities which they regarded as abnormal in the child's conduct.

Parental Attitudes and Relationships.

In common with other behaviour disorders in children, prolonged association with certain types of parents might be responsible for the development of an over-conscientious, meticulous, self-critical child, due to imitation. Over-

insistence on rigidity of toilet training, feeding and sleeping, table manners, being good, honest, truthful, implied pressure and anticipated compliance from an immature child on the part of the mother. Since conformity meant the enjoyment of maternal affection and approval, the loss of which he dared not risk, the child lacked spontaneity, became anxious in his choice of response, and later might find a refuge in ritual. If that relieved his anxiety and satisfied his needs, it might become dominant and permanent in his pattern of behaviour. The more essential did that ritual become when his relationship to his mother was jeopardized by the appearance of a rival sibling. Over-emphasis by the parent on "being good" could stimulate guilty attitudes and anxiety when conformity to standards was difficult or distasteful. Activities with a sexual quality could be a source of guilt, especially if undue stress had been attached to any such practice by the parent. Self-reproach and self-punishment, with protective ritual, especially in the pre-adolescent and adolescent, were typical responses.

Supportive mechanisms, propitiatory actions and thoughts, might develop with the child's inability to establish a warm reciprocal relationship with the parents. Withdrawal of their affection, temporary or permanent rejection, parental disapproval, produced anxiety and insecurity, with the revival and reinforcement of immature techniques and rituals.

Prolonged illness and operations as an infant, separation from the mother, unpredictable and bewildering responses on her part, increased a child's anxiety and predisposed him to certain infantile defence mechanisms.

Anxiety followed by aggression could develop further when the child's relationship to its mother was challenged by another sibling. Hostility developed towards both mother and rival. Later the child might try to be extremely good, failed to achieve what he desired, and fell back on some supportive mechanisms, with his initial disturbance still unresolved. Erotic drives might be displaced similarly from one object to another, so that he could either perform or refrain from performing a certain action.

In those instances, personality patterns could develop to avoid feelings of guilt, with the observance of rituals—compulsions as safeguards against possible risks.

Standards concerned with cleanliness, appearance, conformity to prevailing modes of dress and deportment might be regarded as highly essential and followed meticulously. The need for absolute security and certainty led to the adoption of rigid rules regarding behaviour. The more circumscribed and reliable his regime, the more secure the child felt in reducing unfamiliar situations to a minimum.

Dr. Sebire said that many of the children seen at her clinic with an obsessional pattern had a relatively high standard of intelligence. The last four patients who could represent a sampling had intelligence quotients of 140, 122, 121 and 100 respectively, their ages ranging from fifteen years to six years. The three eldest, and incidentally the brightest, were referred with their obsessional characteristics well organized, elaborated and formulated. The youngest showed symptoms more phobic in character, but presented a pattern which was threatening to develop into compulsive rituals and observances. He made a remarkable response to sessions of play therapy.

Psychotherapy.

Dr. Sebire said that to those who, like herself, were inexperienced in psychoanalytical technique and practice, psychotherapy presented difficulties. Their chief substitute lay in intensive and detailed history taking, with emphasis on the quality of family relationships and parental attitudes. Children in the later latency period and early adolescence could generally describe with remarkable insight their conscious feelings and resultant behaviour. They responded to an opportunity to make admissions when free from parental pressure and criticism regarding the cessation or curtailment of persistent practices. As in all the neuroses, one could assume that psychotherapy was already in train with the first interview.

In the adolescent, morbid preoccupation with sex, religion, immortality and eternity might present fertile fields for rumination, as well as for psychiatric exploration. That was often symptomatic of a transitional period when abstract concepts came up for consideration, or the recall of earlier indiscretions, default and wrong-doing provided a focal point for self-criticism and obsessional thinking. Often tension was relieved by the psychiatrist's admission of a struggle with similar doubts and anxiety during the same period.

Supportive therapy through interviews continued for some time, and the patient's acceptance, at least intellectually, of the explanation of his use of particular mechanisms, together with interpretation in terms of his own strivings and family relationships, could ease the conflict in many instances. The formation of substitute interests and activities with groups encouraged socialization and limited solitary concern and individual stereotyped practices. Achievement in that direction often modified feeling, thinking and functioning at a previous immature level.

Failure was often associated with lack of cooperation in the parents, probably inadequate or too demanding in the first instance, and later exasperated, impatient and often vindictive. One was sometimes in the unfortunate position of attempting to justify impossible parents to a child. Dr. Sebire wondered whether one should regard them as part of a reality situation to which the patient was required to adjust himself, as to any other liability or handicap. She said, in conclusion, that if predisposing patterns of behaviour were recognized sufficiently early, then the emphasis should be on parent education in training and management. Parents needed encouragement in tempering their attitudes, demands and prohibitions, in easing their standards, in limiting blame and criticism, and in giving a more generous demonstration of affection and approval.

Psychotherapy of Obsessional Neurosis.

ANDREW PETO discussed the psychotherapy of obsessional neurosis. He said that psychoanalytical treatment was based on the phenomenon of transference. That term had been coined by Freud to describe all reactions that were provoked in the patient in the course of the treatment, and were directed towards the analyst. Freud had discovered that analytical therapy—as any kind of psychotherapy—impelled the patient to build up a complex emotional relation to the therapist. Thus all positive and negative feelings that were at the patient's disposal—love as well as hate—turned towards the analyst. The analyst's task was to "work through" those attitudes that were "transferred" on to him. They represented the patient's main and basic emotional attitudes, which had developed throughout the course of his life. That emotional register embraced not only the adult patterns, but those of childhood and adolescence—in other words, the decisive reaction that had built up the patient's character.

The positive (loving) and negative (hateful) transference gave the analyst the opportunity to understand the patient's instinctual and ego development, and revealed the causes of the patient's faulty adaptations, which had led to his symptoms. As the patient relived his past and present object relations in the transference, the analyst—by persistent interpretations of the patient's attitudes—confronted the latter with his inadequate reactions to his past and present life. The patient repeated in his relation to the analyst all phases and symptoms of his neurosis—he developed what Freud called the transference neurosis.

The positive trends of that transference neurosis were partly helpful in the therapy, because they tied the patient to the analyst and enabled the patient to bear the tension of the analytical situation. Nevertheless the same positive feelings, together with the negative ones, represented what was called the "resistance", because they impelled the patient to react and feel in his old neurotic ways. In other words, the patient attempted to pull the analyst into the faulty structure of his neurosis. Consistent analysis of the resistances was one of the basic principles of analytical technique, which aimed at the reshifting of the old attitudes.

Freud had assumed that only those syndromes were analytically approachable in which the patient was able to develop a transference neurosis. Obsessional neurosis was one of the syndromes that he ranked among the analytically curable diseases. In one of his classical case histories, that of the so-called rat-man, he had discussed the vicissitudes and cure of such a patient.

Since transference was part and parcel of the analytical approach, therapy had to be considered from that viewpoint. Andrew Peto said that there he had to stress that the difficulties of building a steady and reliable transference made the cure a precarious task with obsessional neurotics. The obsessional's ability to love and to hate in a realistic and mature way was extremely limited. The objects of his instinctual drives and of his emotions in the external world were figureheads, projected shadows of internalized conflicts. The classical symptoms themselves indicated that detachment from reality—he referred to doubt, ritual, guilt, magic of words and gestures, oscillation between contrary thought sequences. Those dynamics might sometimes force the patient into a pseudo-existence.

Analytical research had shown that those symptomatic manifestations represented regression to a developmental stage at which aggression, ambivalence and consequent feeling of guilt prevailed. The symptoms meant defence against the permanent impact of those forces. The obsessional tried to give up the object relations of his childhood and to avoid any new ones in reality, so as to escape from those—for him very effective—phantasies of destruction, revenge and guilt.

The atmosphere of the analytical situation and the consistent interpretations of the analyst forced the patient in the direction of taking up that which was extremely dreaded by him—towards feeling emotions, mostly hate, with all their phantasied consequences. The patient had to face the alternative of the following two solutions: (i) Yielding to his positive emotions towards the analyst. For the sake of that positive transference and with the healthy parts of his ego he dared to face his parallel upsurging hate and the phantasied consequent revenge of the parental imagos that had been projected on the analyst in the transference. In other words, he released the repressed storms of his childhood that had been stifled into the faulty adaptations, represented by his symptoms. (ii) Taking refuge in the old defences and inserting the analyst in the system of doubt, word magic or ritual. A variation of that solution was the discontinuation of analysis through the inability to cope with the anxiety that was conjured up by the revival of the pathogenic conflicts in the transference situation.

Andrew Peto recounted a case of successful analysis. He said that the male patient's main obsession was the counting of coitus movements during intercourse. The outstanding traumatic events that were important for understanding the dynamics of the symptom were the following. He had had a brutal father, who was a teacher of mathematics and later taught the boy at school. The mother died of puerperal sepsis. The child had been brought up by a young aunt, whom the father married when the boy was six years old. The symptom had—among others—the contrary double meaning: "I defy my father and indulge in incestuous intercourse with my mother"; counting meant identification with the father—that was to say, taking his place at the mother. The reversing meaning was: "My father, my superego, punishes me so that I should not enjoy, or because I enjoy, intercourse. I have to count, to rack my brain." Consistent interpretations, which led through emotional storms of homosexual transference and wild hate, resulted in dissolution of the obsessional symptom, which was replaced by a conversion hysterical symptom, a feeling of hotness in the penis. It meant a more mature and more approachable neurotic solution of his incestuous wishes, and its analysis was successful. The patient failed to pull the analyst into his emotionally isolated system of empty counting, analysis forced him to experience love and hate in what Freud called "the heat of transference". The breakdown of his obsessional defences effected a regrouping of his emotional relations. His obsessional defence against the analyst could be described in the following way. In the same way as his sexual life was isolated from emotions, and that isolation resulted in the counting of the coitus movements, so he tried to keep emotional relations away from the analyst in his treatment. He had drawn up a balance of the analyst's good and bad qualities. Those were never decisive and had to be considered coolly—as he said—since emotions always spoiled human relations. The tension of the analytical situation provoked such an impact of impulses of all kind that his obsessional pondering broke down, and he had to face the analyst in the genuine storm of his instinctual drives.

As a contrast, Andrew Peto quoted another case in which analysis failed. The young man was the typical representative of the obsessional with the cleanliness problem. He was in permanent anguish lest his trousers might have been soiled with urine, urethral secretion or sperma. Continuous checking, doubt in its effectiveness, desperate fear of people's noticing the possible stains, problems of guilt and elaborate activities to clean and eventually throw the trousers into the incinerator, were the main manifest symptoms. Analytical deciphering of the symptom had shown that it meant two opposing trends: (i) different layers of incestuous desires and death wishes towards the father; (ii) as a repercussion, shame, guilt and self-destruction. The gained insight disturbed the patient extremely and increased his feeling of guilt. As a defence he tried to consider the analytically gained material as an interesting scientific topic and involved the analyst into his system of doubt. He continuously weighed up whether analysis offered more dirt or more intellectual profit. At last his obsessional intellectualizing and his isolation defences were near a breaking point under the weight of transference. He

started to realize genuine hate against the analyst, and that revived the violent hate of his childhood against his father. After the agonies of a fight between his guilt and the healthy part of his ego that tied him to the analyst and to treatment, the former won the upper hand and he broke off the treatment under some trifling pretext, after having discussed it and confessed that it was a pretext only.

Andrew Peto said that analytical theory assumed that obsession mainly expressed unconscious regression to the mental dynamics of an early stage of development, that prevailing in the second year of life. Thus, as had been shown in the first case he had quoted, that regression of development had to be made up and the patient might go through a stage of hysteria symptoms which represented, according to analytical theory, a more advanced stage of faulty infantile development. Those disintegrative changes in the patient's personality were accompanied, in many patients, by confusional states of different degree. Those confusional states during treatment indicated in most cases that a desirable disintegration of the ego had occurred. It might be limited to the sessions or to a single session, it might stretch over a period of several hours or days. It never hampered the patient from carrying out his usual activities. Those confusions marked the milestones of the piecemeal destruction of the obsessional defence mechanisms. They indicated that the ego had been flooded over by emotions that were hitherto isolated from conscious realization. They occurred in what Freud called "the heat of transference in which the old rigid patterns become melted". Thus an opportunity was given to the patient of readjusting his real relations on the model of his changing emotional attitudes towards the analyst. There were cases in which the obsessional mechanism covered a psychotic process which was coped with in that way. It went without saying that such a diagnosis definitely changed the whole approach, and the earlier it was discovered the smaller the harm that could be done by such treatment. Though all patients with severe obsessional neurosis showed depressive and paranoid traits, the exclusion of psychosis could be made after a few sessions of the usual trial period. If a diagnosis of psychosis was made, then the analyst dismissed the patient or continued the analysis on the basis of his knowledge of a latent psychosis, taking into consideration all the advantages and disadvantages of such a procedure.

Andrew Peto finally mentioned the therapeutic results that he had seen in his own practice. In all cases the symptoms had become manifest before the age of ten years. In all cases taken into consideration the disease caused a definite or total limitation of social or professional activities. What could be called a total restoration of health in every respect was achieved in 40% of the subjects, those treated for three to four years. About 20% showed a definite improvement implying the cessation of manifest symptoms, but persistence to some extent of the rigidity in the emotional sphere; 40% interrupted the treatment during a period of transference crisis or proved to be incurable though they clung to the analysis.

The Treatment of the Obsessive Patient.

DR. GUY A. LAWRENCE discussed the treatment of the obsessive patient. He said that at Broughton Hall Psychiatric Clinic a large number of patients with functional nerve disorders passed through each year, and one was struck forcibly by certain varying difficulties present in the treatment of such conditions. Consideration might first be given to the simple anxiety neuroses, which frequently responded well to the various treatments, and then to the most numerous group, that of the anxiety hysterias; it was in that group that the highest percentage of cures was found. Then in the hysteria group there was a slightly less favourable number of patients discharged as cured. In a consideration of the cases of anxiety hysteria in which phobias were being shown, greater difficulties at once appeared. As Stekel had stated, the anxiety became a phobia when, so to speak, it crystallized in a definite complex; but the patient had not freed himself from anxiety through the phobia, he had only converted the manifest anxiety into a hidden anxiety, and he stood in constant readiness for anxiety. The prognosis in those cases was worse when more than one phobia presented in one person, or when the symptoms passed into a full state of obsessive-compulsive neurosis. In the latter condition the greatest difficulty was met with in successfully treating the patient. If the obsessions were strongly developed, or if they had been present for a long time, then in fact cure was frequently impossible. However, some more or less reasonable remission of severity of symptoms was often possible, by which the sufferer could resume his usual occupation for a

time before seeking more treatment. Many patients remained unrelieved and often passed into a full psychotic illness and had to undergo certification. Some of the sufferers who had been relieved and whose symptoms recurred frequently sought relief in suicide. Dr. Lawrance said that it would be of interest to review the results obtained in the last 50 patients with obsessional neurosis treated, and to compare those with the results in the first 50 patients with the same illness treated at Broughton Hall Psychiatric Clinic from its opening in 1922. The facts disclosed would be somewhat depressing, as there was so little improvement in the results.

Dr. Lawrance said that if the Freudian view was accepted that the obsessive symptoms were due to a repression of libido from the fear and guilt of childhood's sexual adventures, or what passed for them, with the later attachment of that libido to entirely different objects or thoughts in the conscious, it was noted that sex incidents could be disclosed in nearly all the patients. Those incidents had occurred before the age of twelve years; they consisted of masturbation in 35 cases, sex assault in nine cases, sex play in 19 cases and perversions in five cases. The knowledge of those happenings did not aid the patients, who attached no importance to them in connexion with their illness, and, of course, there was no emotional response in regard to them.

Dr. Lawrance then mentioned the types of therapy used. He said that electroconvulsive therapy was used in 40 cases (if depression was present), carbon dioxide and oxygen therapy in five cases (if anxiety was present), "Somnifaine" therapy in two cases (if restlessness was present), insulin coma in three cases (if the condition was of schizoid type), and narcoanalysis in seven cases. Of the first 50 patients treated at Broughton Hall (1922 onwards), nine had recovered, 29 had been relieved and the condition of 12 had not improved. Of the second series of 50 patients (1950 onwards), 10 had recovered, 30 had been relieved and the condition of 10 had not improved. Of the first series of 50, the average stay in hospital was 3.9 months; of the second series, the average stay in hospital was 3.25 months. It was obvious that modern dynamic therapies had not been of much help to the obsessive sufferer. The only gain was represented by a shortening of the average stay in hospital by 0.65 month, and that might even be accounted for by the greater need of hospital beds in the present days of overcrowding, compared with the less tense situation of thirty years ago. It would seem that prolonged psychotherapy conducted by a skilled analyst was necessary, and it would be interesting to hear the views expressed later on at the meeting. Dr. Lawrance said that recently a doctor from England had informed him that in a few cases of severe obsessive illness, success had been obtained at the Mandesley Clinic by the performance of leucotomy; but he (Dr. Lawrance) had had no personal observation of that type of treatment.

Dr. Lawrance went on to say that Dr. Eliot-Slater and Dr. William Sargent, in a discussion held by the Section of Psychiatry of the Royal Society of Medicine in 1950, had said that therapy in the obsessional neuroses was at least as confused as in any other mental disease. In the first place, under insulin therapy, the patient felt a little better while in the semi-stuporose state, but afterwards was worse. Secondly, under sleep treatment he was likely to become worse and more confused, and that confusion could continue after the drug withdrawal, certification might be necessary, and in any case well-established obsessional behaviour patterns were not touched by the therapy. Thirdly, electroconvulsive therapy did not relieve the tensions, and anxiety had flared up after its use. Fourthly, only patients whose obsessions were associated with schizophrenia were benefited by insulin coma therapy. In the fifth place, Eliot-Slater and Sargent felt that leucotomy gave better results in obsessional neuroses than in any other conditions in which the method was used. Monosymptomatic patients responded well to it, and age was no bar to the operation, as a woman, aged seventy-two years, whose obsession was twenty years old, had been cured.

The Treatment of Obsessive States.

DR. A. T. EDWARDS, in a paper on the treatment of obsessive states, said that the main characteristics of the obsessive state were the compulsive tendencies to think in certain ways, frequently with a resulting tendency to act in certain ways. Often the state was described as a tendency to compulsive ideas, but Freud had pointed out that it was much more correct to speak of obsessive thinking than of obsessive ideas. The majority of patients with obsessive states had shown a tendency to that type of thinking from an early age, in childhood often disguised as "magical

thinking"; they must say their prayers a certain number of times or some awful fate would overtake their mother; they must touch each lamppost on the way to school, otherwise they would fall in their examinations. That obsessive type of thinking often continued throughout adult life without ever becoming anxiety-laden, and the individual was surprised to learn accidentally that it was not an experience common to everyone. Under certain circumstances, however, the obsessive thinking became laden with conscious anxiety, and when that occurred there was an exacerbation of the condition, so that the obsessive acts and obsessive thinking became increasingly emphasized with secondary increase in anxiety, and the total condition often became completely disabling. That disability appeared in three conditions. Firstly, it appeared as part of a depressive reaction—either a reactive depression or in the so-called endogenous types—and occasionally the obsessive condition was made manifest in involutional melancholia. Secondly, it might become accentuated by the development of an anxiety state; probably that was more likely to occur when there was a true anxiety neurosis. Thirdly, there was the classical obsessional neurosis in which the underlying obsessive state became anxiety laden either as the result of some environmental factor, or, unfortunately, for no obvious reason. The treatment of the condition was dependent on a recognition of the type of reaction that existed. If a patient had been happy with his obsessive rituals until the development of a depressive state, and if with the clearing up of the depressive state he could again be made happy with his rituals, the policy of *primum non nocere* was to be observed. The eradication of obsessive thinking by any form of treatment was a long and arduous task, and, unfortunately, frequently fruitless. In those cases treatment was to be limited to the depressive condition—and that might need electrostimulation therapy, drug therapy, and/or psychotherapy.

Dr. Edwards went on to say that obsessive states that had been accentuated by an anxiety state were to be treated likewise, but when the obsessive state was mixed with an anxiety neurosis as a mixed psychoneurosis, treatment generally needed to be directed towards both conditions. There, and in the pure obsessional neurosis, in view of the accepted psychodynamics of the latter condition, psychoanalysis was the treatment of choice. However, material considerations limited that treatment to a relatively few patients; the length of treatment, the lack of trained analysts coupled with the large number of patients available for the treatment, financial considerations and the need to enable the patient to return to his job in as short a time as possible, all considerations made it necessary to seek short cuts in treatment. Drug treatment was at times of assistance in enabling the patient to carry on during psychotherapeutic treatment, especially the combination of a barbiturate with amphetamine. In a few cases electrostimulation treatment so far dispersed the accompanying depression, anxiety and tension as to have a definite place in treatment, and paradoxically, although it lessened anxiety, it also tended to make the patient more responsive to psychotherapy—probably on account of the speed with which a patient developed transference during those physical treatments. However, all patients needed some type of psychotherapy, and the type would, of course, depend to a large extent upon the personality of the psychotherapist. Obsessional states were notoriously and admittedly difficult. Frequently one had to be content if one could enable the patient to "live with" his neurosis—to reduce the accompanying anxiety and depression, and to bring the obsessive ritual within reasonable limits. In occasional favourable cases one was completely successful; those cases, in his experience, had been cases in which the obsessively disabling ritual had developed in response to some guilt-provoking episode that had reawakened the original traumatic guilt and anxiety. Conscious reawakening of the secondary guilt-laden memories, with insight into their importance, and reeducation directed towards the abolition of the magical type of thinking were important factors in success. No matter what form of psychotherapy was chosen, those cases were never promising. The road was long and one had to "try often and be content with small gains". Probably no other condition so exposed the three limitations with which they were so often faced—the limitations of psychiatry, the limitations of the psychiatrist and the limitations of the patient. Finally, when all other treatment had failed, when the condition was disabling and when there was a great deal of tension and anxiety, leucotomy was at times indicated. That was a confession of failure, but it did at times make life livable for the patient on a reasonable level of happiness and adjustment.

Discussion.

DR. W. S. DAWSON, in opening the discussion, said that he was still in some uncertainty as to what really constituted an obsessional neurosis. Dr. Swanton had referred to the theorizing about their illness exhibited by some obsessionals, but the tendency to rationalize about symptoms was not a feature peculiar to obsessionals. Dr. Dawson felt that too much attention had been paid to the study of symptoms, to the neglect of their basal setting. Dr. Swanton had mentioned a possible physiological foundation, the existence of a circuit of impulses which might be broken by such therapeutic procedures as leucotomy. Dr. Dawson suggested that the ambivalence shown by obsessionals had a parallel in reciprocal innervation. But the linkage of the Hamletian "To be or not to be" with any neurophysiological mechanism was still very slender. It was of interest to recall that obsessional ideas and actions had appeared as sequels to epidemic encephalitis in patients who had not previously exhibited such symptoms. Dr. Seibre had outlined the sort of approach she made to maladjustments in childhood—an approach which did not seem to be so much directed towards particular symptoms as towards the social, domestic or parental situations in which the child was set; Dr. Dawson considered that that approach was all to the good, and indeed he thought that some of the cases mentioned by Dr. Seibre might equally well be termed anxiety cases. But neurosis in childhood was less formalized than in adult life. Dr. Peto had described the handling of the transference in obsessionals, working through the patient's attitudes towards the analyst who stood in *loco parentis* (or *societatis*), a procedure to which the obsessional offered considerable resistance. A significant number of successes was claimed, sometimes after three or four years of analysis. Not being an analyst, Dr. Dawson felt diffident about offering comment; but he had often wondered if apparent obsessionals who improved under analysis and indeed other treatments were not really depressives with obsessional symptoms. He wished that the opening speakers had made more reference to some of the settings in which obsessional symptoms appeared, especially depressive and schizophrenic states. Dr. Lawrance, after a comparison between series of patients treated at Broughton Hall in the years before and after the introduction of physical methods, had come to the conclusion that the latter, electroconvulsive therapy and oxygen and carbon dioxide narcosis were not strikingly effective in obsessional states. Dr. Edwards had supported other speakers in finding the verbalizing rigid type of obsessional fairly intractable, but had given instances of greater success with other types, even with superficial psychotherapy. Dr. Dawson suggested that on the whole more success was obtained by both physical and psychological therapy when the patient displayed a degree of emotional tension, and in such cases in his experience leucotomy had been of distinct benefit after other methods had failed. The lack of emotion in some ideational obsessives reminded one of the emotional flattening of some chronic schizophrenics.

In response to a question by Andrew Peto about a possible relationship between anal-erotic manifestations and a neurophysiological basis, Dr. Dawson said that it was rather like the old problem of the hen and the egg. Which came first, a physical instability, or certain infantile experiences giving rise to anal-erotism and then or simultaneously to pathologically intermittent abnormal bowel habits? Dr. Dawson said that the questioner had explained that the transference situation could be carried back only as far as the anal-erotic stage. But research along different lines might provide some other explanation. In conclusion, Dr. Dawson paid a tribute to the useful viewpoints presented by the opening speakers.

DR. J. A. NUFFIELD said that in his own limited experience with obsessional cases he had been struck by one or two facts which might be worth mentioning at that stage. The first was the extreme aggressiveness latent in those people, and the difficulty in mobilizing that aggressiveness in the course of therapy. The original object of their hatred had invariably been the mother, even in the case of men, and unless one could relate that hostility to the mother, therapy did not appear to be going on. The second fact was the ease with which active sexual trauma in the past was discovered, as Dr. Lawrance had mentioned earlier. They seemed to be what Freud had called "screen memories", and their discovery brought about no therapeutic result whatsoever. That, of course, was very frustrating to the therapist. Dr. Nuffield said that he supported Dr. Edwards's idea of two distinct groups of obsessionals. The first were the "verbalizers", full of rituals, and possibly not very much disturbed emotionally. They appeared so resistant to all forms of therapy that one wondered whether they were

really meant to be treated. The other group was the disturbed group, with anxiety or depression as prominent accompaniments. It seemed that psychotherapy could do much for them.

DR. I. A. LISTWAN asked two questions. The first was, what was the clinical proof of cure in obsessional neurosis? He said that some people relapsed frequently even after a long period, and even if their personality adjustment was considered perfect or nearly perfect by the analyst or the psychotherapist. The second question was, what were the indications for treatment in obsessional neurosis, with special reference to the so-called monosymptomatic cases? Sometimes those patients were happy with their symptoms, and usually trouble started when the therapist began to dig too deeply. Dr. Listwan said that those two questions were specially important in every-day "minor" psychiatry, with the large turnover of patients who were mostly without funds for analysis. In those cases analysts would like to know whether they had to start, and if they had to start, when to stop.

Dr. Winn, in closing the discussion, said that the consensus of opinion appeared to be that psychoanalysis was impracticable for the majority of patients because of its long duration. However, he thought that, in addition to its therapeutic effects, psychoanalysis was an instrument of research, which by revealing the causes of obsessional neurosis was throwing light on the nature of thought itself.

Out of the Past.

In this column will be published from time to time extracts, taken from medical journals, newspapers, official and historical records, diaries and so on, dealing with events connected with the early medical history of Australia.

FOOD FOR PRISONERS.¹

Colonial Secretary's Office,
Sydney,
22nd March, 1855.

The Medical Adviser to the Government.

Sir,

With reference to your report of 24th ultimo concerning certain proposed alterations in the dietary of the Parramatta Gaol, I am directed to inform you that the Governor-General concurs with you in your opinion of the advisability of an issue of vegetables to the prisoners, but His Excellency cannot think that $\frac{1}{2}$ lb. of vegetables should be given in addition to a ration of 1 lb. of meat and $1\frac{1}{2}$ lb. of bread and meal and 1 oz. of sugar as this would give to each of the prisoners $\frac{3}{4}$ lb. of solid food per diem, which is more than he ought to have.

I have, etc.,

W. ELYARD.

Correspondence.

SUTURE OF THE CONJOINED TENDON TO THE INGUINAL LIGAMENT IN THE BASSINI OPERATION.

SIR: A number of surgeons still seem to be using an extraordinary method of stitching the conjoined tendon to inguinal ligament in Bassini repairs of inguinal hernia. A series of separate sutures is put in, but none is tied till all have been inserted. Each thread passes backwards through the conjoined tendon, across to inguinal ligament, into and back out from inguinal ligament, then through conjoined tendon again from behind, in a situation usually fairly close to the first bite. All sutures are then usually firmly tied.

This horrible procedure drags the few fibres of muscle which each stitch ensnares down to the inguinal ligament under tension and leaves the free edge of conjoined tendon at a loose end, wasting perhaps an inch of precious muscle and strangulating the few fibres (if any) by which each stitch grips the conjoined tendon. Some of them must surely cut out and contribute to the recurrence rate after herniorrhaphy.

¹ From the original in the Mitchell Library, Sydney.

A simple continuous suture, the same as everybody uses for closing the peritoneum in abdominal incisions (an even more important closure), is surely better. A "strong" closure is not necessary, as most authorities agree that the conjoined tendon should not be put under tension. If it will not come easily alongside the inguinal ligament, some other procedure should be used.

Finally, the interrupted suture described is unsuitable where the fibres in the structures united run parallel to the wound. It is more suitable when fibres run at right angles to the wound.

Yours, etc.,

J. WOOLNOUGH.

35 Oxford Street,
Epping,
New South Wales.
February 28, 1953.

not give rise to any of the difficulties or complications which may follow its performance in the Hofmeister operation.

Whilst agreeing with Mr. Rundle that it is a sound policy to learn and practise a standard technique, and have it as nearly foolproof as possible, it is certainly not the ideal policy, as it involves too often the fitting of the patient and his disease to the operation. In whatever field he may practise, a specialist surgeon's technique should be such that he is not confined to one procedure, but is able to perform whatever operation or modification is indicated in the given case. Thus he fits the operation to the patient, rather than vice versa.

Yours, etc.,

ANTHONY KELLY.

33 Collins Street,
Melbourne,
March 5, 1953.

SURGICAL ASPECTS OF THE TREATMENT OF PEPTIC ULCER.

SIR: In a review of the above subject by Mr. F. F. Rundle given at Orange (MED. J. AUSTRALIA, February 28, 1953), I was rather surprised that there was no reference whatsoever to the Billroth I type of operation. As indicated by Dr. J. H. Priestley during the discussion, this type of operation is beginning, at last, to enjoy a well-earned rise in popularity.

The operation can be performed in practically all cases in which the first part of the duodenum is normally mobile; that is, in nearly all gastric ulcers, and in even a few duodenal ulcers. However, it is unsuitable for the majority of duodenal ulcers coming to operation; and for them the Hofmeister-Pólya operation as described must be employed. Being a more "physiological" procedure the convalescence is smoother after the Billroth I type, and the incidence of post-operative troubles both early and late is lower than with the "standard" operation. Having mastered the technique, there is no difficulty whatever in removing an adequate amount of stomach, including the whole of the lesser curvature: in many ways a desired feature which does

Australian Medical Board Proceedings.

VICTORIA.

THE following letter has been received from the Secretary of the Medical Board of Victoria.

SIR: At a recent meeting of the Medical Board of Victoria a medical practitioner was called upon to give an explanation of the circumstances surrounding a conviction recorded against him in a Civil Court arising out of a charge of driving a motor-car whilst under the influence of intoxicating liquor.

The decision of the Civil Court was: "Fined thirty pounds (£30) in default distress. Order that defendant's driver's licence be cancelled."

Upon hearing the doctor's explanation of the case the Board informed him that it was not intended to record any penalty against him. However, the Board further informed the doctor that it has power under the Victoria Medical Act to reprimand, suspend the registration, or erase the name of a medical practitioner who has been convicted of a mis-

DISEASES NOTIFIED IN EACH STATE AND TERRITORY OF AUSTRALIA FOR THE WEEK ENDED FEBRUARY 28, 1953.¹

Disease.	New South Wales.	Victoria.	Queensland.	South Australia.	Western Australia.	Tasmania.	Northern Territory.	Australian Capital Territory.	Australia.
Acute Rheumatism	1	1(1)	2
Amoebiasis
Ancylostomiasis	4(1)	4
Anthrax
Bilharziasis
Brucellosis
Cholera
Chorea (St. Vitus)
Dengue
Diarrhoea (Infantile)	2(2)	3(2)	7(4)	1	4	..	17
Diphtheria	9(6)	7(6)	2(1)	..	2(2)	20
Dysentery (Bacillary)	2(2)	1	4(4)	5(4)	1	13
Encephalitis	1	1	2
Filariasis
Homologous Serum Jaundice
Hydatid
Infective Hepatitis	10(2)	12(6)	..	1	..	23
Lead Poisoning	1	1
Leprosy
Leptospirosis	1	1
Malaria
Meningococcal Infection	5(4)	1(1)	..	4	6
Ophthalmia	4
Ornithosis
Paratyphoid
Plague
Pollomyelitis	24(10)	12(4)	2	20(13)	..	3(1)	61
Puerperal Fever	3	..	3
Rubella	23(16)	2(2)	25
Salmonella Infection	3(3)	3
Scarlet Fever	15(10)	45(31)	1(1)	2(1)	3(3)	1	67
Smallpox
Tetanus	1(1)	1
Trachoma
Trichinosis
Tuberculosis	23(16)	21(14)	10(6)	7(3)	7(6)	4(3)	1	..	73
Typhoid Fever	1(1)	1
Typhus (Flea-, Mite- and Tick-borne)	1(1)	1
Typhus (Louse-borne)
Yellow Fever

¹ Figures in parentheses are those for the metropolitan area.

demeanour in a Civil Court, and warned him that any similar case in which he might be involved in the future would have serious consequences for him.

By direction, I have to inform you that the Board would be pleased if you would give publicity to the matter in THE MEDICAL JOURNAL OF AUSTRALIA as it considers that it is one that should be brought under the notice of medical practitioners.

Yours, etc.,

JOHN F. McCORKILL,
Secretary.

295 Queen Street,
Melbourne,
March 16, 1953.

Public Health.

DIAGNOSTIC CONSULTANTS IN POLIOMYELITIS IN NEW SOUTH WALES.

THE following revised list of diagnostic consultants in poliomyelitis in New South Wales is published at the request of the Secretary of the Hospitals Commission of New South Wales.

- Dr. Douglas Anderson, 185 Macquarie Street, Sydney. BW 6944, XY 5757.
Dr. I. A. Brodziak, 231 Macquarie Street, Sydney. BW 9910, FU 2005.
Dr. Ethel Byrne, Royal Newcastle Hospital, Newcastle.
Dr. C. A. Clark, 20 Bolton Street, Newcastle.
Dr. M. L. Edwards, 398 Chapel Road, Bankstown. UY 1613.
Dr. Gertrude S. Geikie, 149 Macquarie Street, Sydney. BW 8616, JX 2109.
Dr. Bruce Hall, 185 Macquarie Street, Sydney. BW 9044, XB 5013.
Dr. K. T. Hughes, 135 Macquarie Street, Sydney. BW 6871.
Dr. W. P. MacCallum, 143 Macquarie Street, Sydney. BU 1149, FM 4266.
Dr. P. J. Markell, 149 Macquarie Street, Sydney. BU 4662, FM 4034.
Dr. C. G. McDonald, 143 Macquarie Street, Sydney. BU 2071, FB 3927.
Dr. F. L. Ritchie, 225 Macquarie Street, Sydney. BW 9071, FB 2034.
Dr. A. T. Roberts, 5 Union Street, Newcastle.

Nominations and Elections.

The undermentioned¹ have applied for election as members of the South Australian Branch of the British Medical Association:

- Brown, Ross Alvarez, M.B., B.S., 1950 (Univ. Adelaide), 19 Elm Street, Unley Park, South Australia.
Stobie, Peter James, M.B., B.S., 1952 (Univ. Adelaide), 51 Austral Terrace, Malvern, South Australia.
Ashton, Thomas James, 2 Halsbury Avenue, Kingswood, South Australia.
Torr, Thomas Harold, Bute, South Australia.
Kalinovsky, Peter Victor, Box 192, Barmera, South Australia.
Kalinovsky, Galina Vera, Box 192, Barmera, South Australia.
Basher, Keith Cameel, 29 Swaine Avenue, Rose Park, South Australia.
Brown, Noel John, 17 Elm Street, Unley Park, South Australia.
Murray, Donald Bruce Seymour, Laura, South Australia.
Law-Smith, David Gordon, Box 41, Gawler, South Australia.
Sando, Maurice James Wilson, 28 Collins Street, Collinswood, South Australia.

The undermentioned¹ have been elected as members of the South Australian Branch of the British Medical Association: Downing, Robert Gregory, M.B., B.S., 1952 (Univ. Adelaide); West, John Burnard, M.B., B.S., 1952 (Univ.

¹ Applicants and newly elected members for whom no degrees are shown qualified in December, 1952, at the examination for the degree of M.B., B.S. (Univ. Adelaide), but the degrees had not been conferred at the time when their applications for membership were considered by the Branch Council.

Adelaide); Potts, John Louis, M.B., B.S., 1950 (Univ. Adelaide); Horton, Robert Ralph; Davies, Norma Esther; Tonkin, David Oliver; Harris, Digby Ian; Gillen, Robert Spencer; Marshall, Janette Ruth Watson; Chappell, William Thomas; MacBeth, William Andrew Alexander Greer; Page, Leland Ivor.

Deaths.

THE following deaths have been announced:

GUILFOYLE.—Francis Patrick Guilfoyle, on March 6, 1953, at Melbourne.

DONOVAN.—Francis Gerald Donovan, on March 16, 1953, at Melbourne.

Diary for the Month.

APRIL 7.—New South Wales Branch, B.M.A.: Organization and Science Committee.

APRIL 10.—Queensland Branch, B.M.A.: Council Meeting.

APRIL 13.—Victorian Branch, B.M.A.: Finance Subcommittee.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Medical Secretary, 135 Macquarie Street, Sydney): All contract practice appointments in New South Wales.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federal Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225 Wickham Terrace, Brisbane, B17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178 North Terrace, Adelaide): All Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205 Saint George's Terrace, Perth): Norseman Hospital; all Contract Practice appointments in Western Australia. All government appointments with the exception of those of the Department of Public Health.

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MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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